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Issues Related to the Decontamination of Chemically Contaminated Human Remains

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U.S. Army Medical Research
Institute of Chemical Defense
Aberdeen Proving Ground, MD 21010-5400

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13. ABSTRACT (*Maximum 200 words*)

This report, based on a search of the available literature, addressed issues specific to problems of decontamination of chemically contaminated human remains. Specific areas of concern included: 1) identification of a worst case situation; 2) determining if hypochlorite is the best decontaminant available; 3) determining the optimum pH for hypochlorite use as a decontaminant; 4) determining an optimum concentration of hypochlorite; 5) determining whether there is benefit in using the sodium salt over the calcium salt of hypochlorite; 6) determination/identification of the agent(s) most difficult to decontaminate; 7) estimation of amount of agent to which remains would be exposed; 8) estimation of how much, if any, agent could be found in decontaminated remains that might be hazardous to handlers of the remains subsequent to decontamination; and 9) determining if there are cosmetic effects produced by the decontamination process. Because the Army was proceeding to write doctrine for a specific decontamination procedure, the questions addressed in this report were focused to those issues that were of prime importance to near-term development of a field system for decontamination of chemically contaminated human remains so that the remains could be released to the family of the deceased.

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EXECUTIVE SUMMARY

Many problems are associated with the decontamination of chemically contaminated remains. Of primary importance are the following: (1) Does the decontaminant work? (2) Even after decontamination, is a residual level of agent present, i.e., in depots or pools, that could pose a hazard to someone handling the remains without adequate chemical agent protection? (3) Are any cosmetic effects produced by the decontamination procedures? These and related issues are considered in this report.

A scenario for severe exposure was developed for use in evaluating problems associated with handling chemically contaminated remains. (1) The remains to be decontaminated had been exposed to VX, the agent that is the most difficult to decontaminate. (2) The remains had been exposed to a maximum amount of agent, i.e., by immersion in a puddle of agent (this translates to coverage of the remains by ~200 g of agent). (3) Exposure was prolonged, i.e., up to 24 hours. (4) The procedures for decontamination and handling of the decontaminated remains would be the same as described in the document "Joint procedures for decontamination and disposition of chemically or biologically contaminated human remains," (1991) which establishes for the surface decontamination of agent a ~15-minute period of immersion in a 1.3-m^3 tank containing the decontaminant solution.

In addition to whole-body immersion, another exposure scenario was investigated: the exposure of remains to agent delivered by munitions. Since agent casualties might also sustain conventional injuries, the role of wounds in affecting the amount of agent absorbed by the remains was also considered.

CHEMISTRY OF DECONTAMINATION WITH HYPOCHLORITE

Our analysis indicates that under the conditions and limitations specified above the optimal conditions for use of hypochlorite solution for the decontamination of chemically contaminated remains include (1) the use of the sodium salt, (2) temperature of the decontamination solution maintained at 25°C (for a 15-minute immersion), (3) a concentration of $\geq$ 5%, and (4) pH maintained at $\geq$ 11.0.

Kinetics of Hypochlorite Decontamination

Consideration of the conditions favorable to decomposition of the chemical agents reveals a limited range of conditions that will adequately decontaminate all chemical agents.

Decomposition of VX, which is considered to be the worst-case agent, is favorable at pH < 7, unfavorable at pH 7-10, and again favorable at pH $\geq$ 11.0. At pH 7-10, it has been reported that toxic products are produced.

Decomposition of GB, and by analogy that of GA and GD, is optimized at pH > 7 and discouraged at lower pH values. The lack of rate data for GB in acidic solutions (i.e., pH < 5) is an information gap; nonetheless, acidic decomposition rates for GB are expected to be significantly lower than those in alkaline solution.

Optimal conditions for HD decomposition cannot be currently identified, but HD is known to be readily degraded by both acidic and alkaline hypochlorite.

It is suggested in this report that decontamination of thickened agents would be slower and more difficult than decontamination of unthickened agents. However, other sources have suggested that the small amount of thickener required has little, if any, effect on the availability of agent to the decontaminant; thus little difference in the rate of decontamination would be seen.

The sole condition that is favorable for decomposition of both VX and G agents in hypochlorite solutions is pH > 11.0. HD will also decompose readily at this pH, but at an unknown rate.

Temperature Dependence

It was found that control of temperature and pH is essential for successful decontamination. Any design for field systems should incorporate features which control these parameters, such as appropriate heating and buffering systems. It is anticipated that the pH variation of the decontamination solution will be influenced by the presence of body fluids from the contaminated remains. The magnitude of this influence and the details of the pH variation during actual decontamination require test validation.

Temperature of the bath also determines the optimum time of immersion of the remains, e.g., at 25°C, optimum immersion time in a 5.25% NaOCl solution (pH > 11) is 15 minutes. This immersion time is of great importance to cosmetic effects that might occur.

Formulation

The formulation of the hypochlorite solution does not significantly affect reaction rates. Both sodium and

calcium formulations are readily available. However, practical considerations indicate that sodium hypochlorite solutions are preferable. Calcium formulations result in the formation of precipitates as decontamination proceeds and cannot be buffered adequately. Thus sodium formulations provide efficient decontamination solutions without unwanted practical drawbacks.

Concentration

The concentration of the hypochlorite solution significantly affects the rate of decontamination. Comparison of projected decontamination rates for 5.25% and 0.5% sodium hypochlorite revealed inadequate rates for 0.5% solutions, while the 5.25% solution provided adequate decontamination times.

Hydrolysis

All of the agents considered in this report hydrolyze slowly at neutral pH and would be expected to persist in physiological solutions if no alternative degradative pathways were present. Mustard is only slightly soluble in water, so actual rates are slow; mustard in solution, however, hydrolyzes very rapidly.

HAZARD TO HANDLERS OF REMAINS SUBSEQUENT TO DECONTAMINATION

It is concluded that only one agent, VX, could produce contamination that might pose a hazard to persons handling the remains subsequent to decontamination. Such a hazard would be largely a contact hazard.

Exposure Scenarios

Two exposure scenarios were considered in addressing this issue: the exposure resulted (1) from total immersion in a puddle of agent or (2) from agent delivered by munitions. The role of wounds was also considered.

Fate of Agent in the Body

Using data from animal and human studies, it was concluded that the G agents or HD remaining after decontamination procedures likely would be destroyed adequately by spontaneous or enzymatic hydrolysis and would not pose a problem to handlers of the remains subsequent to decontamination. There is a slight possibility that the G agents of intermediate volatility could reach potentially hazardous levels inside the body bag due to off-gassing of agent that had penetrated into the skin and was not destroyed during the decontamination procedure.

VX, on the other hand, having a low vapor pressure, is thought to be a significant contact hazard to unprotected handlers because it might persist in the remains after decontamination.

It was concluded that all G agents and VX would not be detoxified substantially by reaction with either specific or nonspecific targets in the remains. These sites would be rapidly saturated by the supralethal quantities of agent to which the remains would be exposed. The detoxification of HD, however, would be substantial.

The half-lives in tissue for HD and G agents are measured in minutes, whereas that for VX is measured in hours. This difference is due in part to the existence of enzymes in skin, blood, and other tissues that increase the rate of hydrolysis of the G agents. No such enzymes are thought to exist for VX.

Levels of Active Agent in Remains

Estimated values for active agent in remains were determined for both whole-body immersion and munitions-delivered exposures. A strategy employing human toxicity units (LD_{50}) was developed for the estimation of the levels of active agent present in remains. Values for whole-body immersion are expressed in terms of the percutaneous LD_{50} value. For exposure by immersion, the following LD_{50} values of agent were calculated: VX = 21,600, GD = 592, GB = 130, TVX = 34,560, and TGD = 592. Values for exposure to agent from munitions were VX = 400, GD = 57, GB = 118, TVX = 960, and TGD = 570. These maximal levels of percutaneously absorbed agent are expected to be confined largely to skin tissue and to diminish in a time-dependent manner (due both to detoxification and to diffusion into tissues and fluids).

Estimates were made of maximal levels of active agent in remains after cutaneous exposure. The thickened agents and VX produced the highest levels of contamination. These levels of percutaneously absorbed agent would be expected to be confined to skin tissue and to diminish in a time-dependent manner due to hydrolysis and enzymatic detoxification and to redistribution via diffusion into surrounding tissue.

Role of Wounds

It is anticipated that agent levels will be higher in remains in which the integrity of the skin barrier has been compromised by wounds. It was predicted that the largest increase in agent content under these conditions would be to remains exposed to G agents. The presence of a 100-cm^2 wound was calculated to increase absorption of G agents by ~2000-fold. This compares to an increase for VX-exposed remains of ~18-fold.

COSMETIC EFFECTS ASSOCIATED WITH DECONTAMINATION PROCEDURE(S)

Under the exposure conditions and decontamination procedures being considered, it is likely that cosmetic effects will occur.

Dissolution of Soft Tissues

Soft-tissue dissolution can be expected to be promoted: (1) in remains that have begun to decompose, and possibly in remains that have deep or extensive wounds; and (2) in remains that have been exposed to vesicants. Embalming cannot be successfully performed on remains that undergo extensive soft-tissue dissolution. To minimize tissue solubilization, it is recommended that the pH of the decontaminating solution be maintained as near pH 11.0 as possible and that temperatures be maintained near 5°C particularly during transport of the remains.

Alterations in Appearance

The appearance of the remains will be altered due to discoloration, dehydration, and saponification, which is promoted by the hypochlorite decontamination solution. It is possible that hair will be lost. Successful cosmetic restoration cannot be achieved if these types of cosmetic changes occur in the face or hands.

Attenuation of Germicidal Activity

Although hypochlorite solutions are widely used as disinfectants and germicides, the solutions recommended in this report for decontamination of chemical threat agents may not promote extensive killing of decay-promoting bacteria or pathogens because of their high pH and buffering capacity. If this is the case, remains can be expected to continue to undergo decomposition after decontamination, and embalmers at Mortuary Dover will have to treat chemically decontaminated remains as a potential biohazard until other germicidal procedures are employed.

Reactions with Embalming Chemicals

Although hypochlorite reacts with components in embalming fluids to produce acutely toxic and carcinogenic products, these interactions are not likely to occur under the conditions employed at Mortuary Dover and at most licensed funeral homes.

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ISSUES RELATED TO THE DECONTAMINATION OF CHEMICALLY CONTAMINATED HUMAN REMAINS

1. INTRODUCTION

In July 1992 SAIC was asked to prepare a report addressing issues specific to problems of decontamination of chemically contaminated human remains, based on a search of the available literature. These specific areas of concern were discussed in the early meetings: (1) What is the worst-case exposure scenario? (2) Is hypochlorite the best decontaminant available? (3) What is the optimum pH for hypochlorite use as a decontaminant? (4) What is the optimum concentration for hypochlorite use as a decontaminant? (5) Is there benefit in using the sodium salt rather than the calcium salt of hypochlorite? (6) What are optimum safe levels of decontamination? (7) Is there any possibility that persons handling the remains subsequent to their decontamination might be exposed to toxic levels of agent? (8) Are cosmetic effects produced by the decontamination process?

The Army was proceeding to write doctrine for a specific field system for decontamination of chemically contaminated human remains. For this reason, the questions to be addressed in this report were focused on issues of prime importance to near-term development of decontamination procedures that could be followed by release of the remains for whatever funeral rites are desired by the family of the deceased. The selected areas to be investigated (a modification of the original eight areas of concern) were (1) estimation of the adequacy of hypochlorite as the decontaminant; (2) identification of the agent(s) most difficult to decontaminate; (3) formulation of a worst-case scenario; (4) estimation of the amount of agent to which remains would be exposed; (5) estimation of how much agent, if any, could be present in decontaminated remains and whether this amount might be hazardous to handlers of the remains subsequent to decontamination; and (6) estimation of what cosmetic effects, if any, might result from the decontamination procedures. These issues are addressed in the three sections that follow.

2. CHEMISTRY OF DECONTAMINATION WITH HYPOCHLORITE

2.1 INTRODUCTION

The decontamination of chemically contaminated remains in the field raises a number of issues related to the chemistry of the decontamination process. A fielded decontamination system must effectively destroy all of the agents that could be encountered within the constraints of the expected field conditions. The chemical agents considered in the report are VX, HD, GD, GA, and GB, including thickened HD and GD. Field conditions that will impact the chemistry of decontamination include (1) variable field temperatures, (2) varying degrees of contamination, (3) interfering reactions of body tissues and fluids with decontamination solution, and (4) time limitations for maintaining practical decontamination schedules. Within these limitations, it is desirable to have a single solution that is effective in decontaminating all of the agents under consideration. Aqueous solutions of hypochlorite have been proposed for the decontamination of chemically contaminated remains.

The decontamination process can be viewed as a two-phase process. The first phase involves removal of the agent contaminant from skin while the second phase involves chemical reaction of the removed agent with the decontamination solution. Both phases are critical for successful decontamination: chemical degradation cannot be accomplished without dissolving the agent into the decontamination solution. Further, when a body is removed from the decontamination bath, the residual bath water on the body is nontoxic only if chemical degradation has reduced agent concentrations below threshold toxicity levels.

Removal of agent from skin is governed primarily by agent solubility in the decontamination solution. A number of the anticipated agents are poorly soluble in aqueous hypochlorite solutions, particularly HD, VX, and the thickened agents. For these agents, additional means of removal are required. Providing ultrasonic agitation along with appropriate mixing devices in the fielded decontamination system is recommended to improve removal of insoluble agents.

The chemical kinetics of reaction between agent and hypochlorite governs the chemical degradation of solubilized agent. Studies of the reactions of agents with solutions of hypochlorite have shown that these solutions effectively destroy each of the neat agents under consideration, although detailed rate data are not available for all of the agents, particularly for thickened agents. The available data indicate that hypochlorite solutions are effective surface decontaminants, but that the optimal conditions are agent-dependent.

For example, VX can be decontaminated in both acidic and alkaline hypochlorite, while GB, according to available data (Epstein *et al.*, 1956; Epstein *et al.*, 1955), is decontaminated effectively only in alkaline solution. Further limitations in the range of alkalinity are imposed by the formation of toxic products when VX reacts with hypochlorite in the pH range of 7-10. Alkaline conditions of pH 11 or higher are suitable for decontamination of both VX and GB and are expected to be suitable for HD as well, although rate data for HD are not currently available. The lack of specific rate data for HD, GA, GD, THD, and TGD, as well as for GB in acidic solution, leaves significant information gaps which prevent a determination of optimum solution parameters.

The information at hand suggests that a hypochlorite solution that is an optimal decontaminant for all agents is not available. A compromise between the optimal conditions for decontamination of each agent is predicted to result in conditions that are adequate for decontamination of all agents. On the basis of available rate data and product toxicity, a limited range of solution conditions is suitable: alkaline conditions ($pH \geq 11$) and temperatures ranging from 5°-25°C. Readily available hypochlorite solutions (i.e., 5.25% NaOCl) are effective for use under these conditions.

Given this set of conditions, the effectiveness of a model field decontamination system can be estimated for model exposure scenarios. Simple models can identify the limitations and determine unresolved complications that require test validation in the design of actual field systems.

2.2 GENERAL CHEMICAL INFORMATION

2.2.1 Composition of Hypochlorite Solutions

Aqueous hypochlorite is well known as a mild oxidizing agent and has been widely used for the decontamination of various agents, particularly HD and VX (Yurow, 1981; Yurow and Davis, 1982). The hypochlorite molecule contains a chlorine atom with a formal oxidation state of +1. This is the origin of the oxidizing capacity of hypochlorite, which is driven by the tendency for chlorine atoms to be reduced to a -1 oxidation state. Solutions of hypochlorite can be

obtained from a variety of well-established processes, including electrolysis of seawater, reaction of Cl_2 gas with aqueous sodium hydroxide, and others. The compositions of the resulting solutions vary as a function of pH according to the equilibrium:

$$OCl^- + H_2O \rightarrow HOCl + OH^-$$

From the expression $K_b = K_w/K_a$, and the K_a value of 4×10^{-8} (Epstein *et al.*, 1956; Epstein *et al.*, 1955), the following relationship between the concentrations of solution components can be derived:

$$[OCl^-]/[HOCl] = 4 \times 10^{(pH-8)}$$

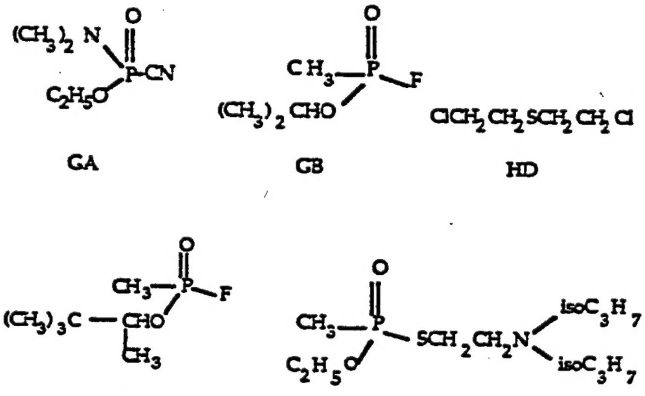
Figure 2.1 shows the ratio of $[OCl^-]$ to [HOCl] as a function of pH. In alkaline solution (pH > 10), essentially all hypochlorite is in the OCl^- form, while in acidic solution (pH < 6), HOCl predominates. In acidic solution, HOCl disproportionates to $Cl_2(aq)$ and HCl. This process is governed by the following equilibrium:

$$HOCl + H^+ + Cl^- = Cl_2(aq) + H_2O \quad K = 2.4 \times 10^3$$

Because the equilibrium constant is large, Cl_2 formation is favored and is promoted by increasing the concentration of HOCl and decreasing the pH. It follows that a significant amount of Cl_2 exists, especially at pH 1-4. Qualitatively, this variation in hypochlorite composition results in different reaction mechanisms and product distributions as a function of pH. This issue has not been extensively characterized in the literature. Nonetheless, the variable composition of hypochlorite solutions determines, to a large extent, the optimal decontamination conditions for each agent.

2.2.2 Molecular Structure and Physical Properties of Chemical Agents

The molecular structures of GA, GB, GD, VX, and HD are shown below.



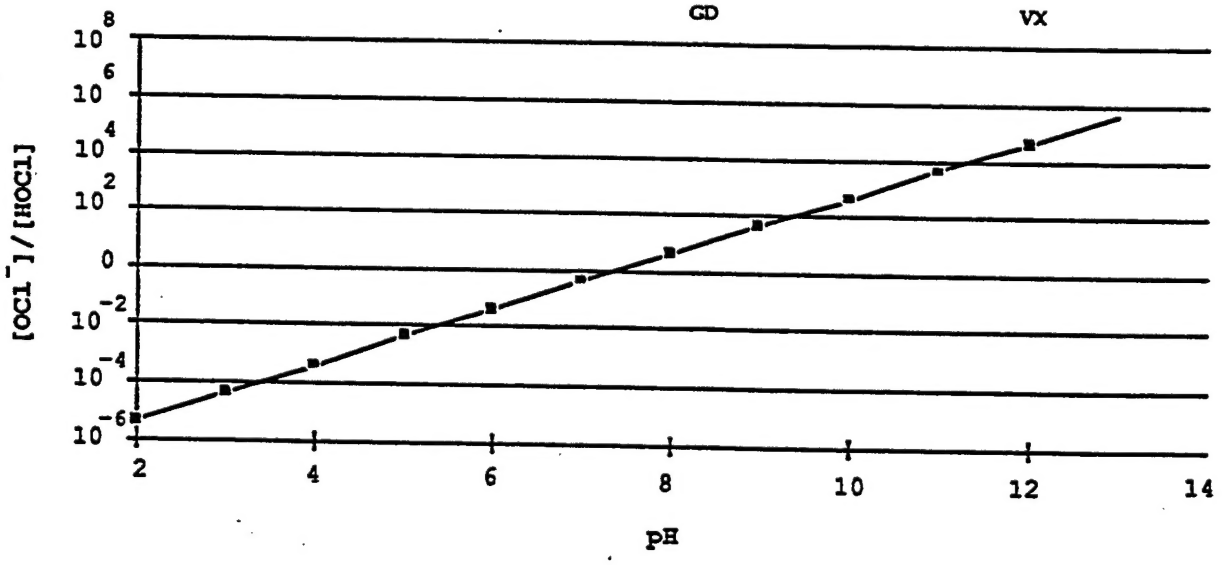


Figure 2.1. Hypochlorite composition as a function of pH.

At 25°C, GA and GB are completely miscible with water, while VX and GD are moderately soluble (30 and 21 g/L, respectively), and HD is sparingly soluble (1 g/L) (Yurow, 1981; Yurow and Davis, 1982; Anderson, 1974). Thickened agents are insoluble as well (Anderson, 1974).

Both HD and VX contain sulfur atoms that are subject to oxidation and are thus particularly vulnerable to degradation by hypochlorite (VX contains an oxidizable nitrogen atom as well). GD, GA, and GB are not readily oxidizable, but are subject to nucleophilic displacement at the phosphonyl phosphorus atom. While hypochlorite is not a particularly strong nucleophile, its reaction with GB is thought to involve a transition state in which the polar hypochlorite ion facilitates attack at the phosphonyl phosphorus by further polarizing the phosphorus-oxygen double bond (Epstein *et al.*, 1956). Structural similarities between GB, GA, and GD suggest that GD should be subject to this mechanism as well, although reaction rates for GD are expected to be somewhat lower than those for GB due to the increased hindrance to nucleophilic approach caused by its pinacolyl group. The hindrance to nucleophilic attack is essentially the same for GA and GB, but decomposition rates for GA are expected to be lower due to the presence of a poorer leaving group (CN^- for GA vs F^- for GB).

Reactions with thickened agents are complicated heterogeneous reactions in which the decontamination solution interacts with a particle of agent-containing polymer. The rates and mechanisms of these reactions are not well understood and no data are available.

Agent persistence is the longevity of its hazard as a toxicant. A rough correlation exists between persistence and other agent physical properties such as vapor pressure and solubility: volatile agents with higher vapor pressures tend to be less persistent than nonvolatile agents with low vapor pressures. While the surface of concern for contaminated remains is skin, the available data for persistence are typically cited for soil or various industrial materials. There may be large differences in agent persistence in different materials because persistence is highly dependent on the means of dispersal (droplet size), the porosity of the contaminated surface, and the solubility of agent in the contaminated material. Extrapolation of agent persistence data from soil or other material to human skin should be done with caution. Lacking directly relevant data to the contrary, here we assume the transferability of the available persistence data to human skin.

An examination of agent vapor pressures and volatilities indicates that agent persistence is ranked as follows: VX > GA > HD > GD > GB (see data in Table 2.1, compiled from Anderson, 1974). The persistence of nerve agents in soil varies with a number of physical and chemical properties of the soils. Agent persistence in soils generally conforms to the same ranking. VX, GA, and GB are persistent for the ranges 2-6 days, 1-5 days, and 0.1-1 day, respectively (Anderson, 1974). HD persistence in soil was described as follows: When sprayed on soil, HD remains vesicant for about 2 weeks; when mixed into soil, HD remains vesicant for more than 3 years (Anderson, 1974). GD was listed as relatively persistent in soil. Persistence listings were cited without a specifying quantitative basis for the values.

Table 2.1. Vapor pressure and volatility of selected agents.

Agent	Vapor Pressure (torr, 25°C)	Volatility (mg/m^3, 25°C)
GA	0.07	610
GB	2.9	22,000
GD	0.40	3,900
VX	0.0007	10.5
HD	0.11	920

2.2.3 Kinetics of Hypochlorite Decontamination

Kinetics studies indicate that each of the agents under consideration (no data were available for thickened agents) can be effectively decontaminated with solutions of hypochlorite. However, the reaction mechanisms which accomplish the degradation and the resulting products vary as a function of pH. Differences in the molecular structures of the agents result in different reactivities under the same conditions. Consequently, optimal solution parameters for decontamination of each agent differ, and all-agent decontamination capability can only be achieved by compromise among the various requirements for agent decontamination.

2.2.3.1 Reaction of GB and Other G Agents with Hypochlorite

Kinetics data indicate that optimal conditions for GB decontamination require an alkaline hypochlorite solution. Studies conducted by Epstein (Epstein *et al.*, 1956, Epstein *et al.*, 1955) reveal the pH dependence of

the GB-hypochlorite reaction. These data (Table 2.2) indicate rates of GB reaction with dilute hypochlorite solutions. The extension of Epstein's data to relevant field conditions requires a three- to four-order-of-magnitude extrapolation in concentration. Reasonable estimates are provided from kinetics theory but require experimental validation. The data indicate a first-order reaction with respect to hypochlorite concentration. Using kinetics theory, the rate constant k at any hypochlorite concentration can be determined if k_{obs} is known at one hypochlorite concentration (provided other solution parameters such as temperature, pH, and GB concentration remain fixed). Assuming a 5.25% NaOCl solution (0.7 M NaOCl), in which the [OCl^-] varies as a function of pH as shown in Figure 2.1, the estimated rate dependence and associated half-lives of GB as a function of pH are shown in Figures 2.2 and 2.3.

Table 2.2. Rate data for GB reaction with hypochlorite at 25°C ([GB] = 2×10^{-4} M).

pH	[OCl^-] $\times 10^{-3}$M	k_{obs} (min^{-1})	$t_{1/2}$ (min)
5	2.02	0.0072	96
6	2.12	0.046	15.1
	0.705	0.016	42.4
7	0.265	0.036	19.2
	0.354	0.0546	12.7
8	0.088	0.04	17.4
	0.132	0.06	11.6
	0.176	0.084	8.3
9	0.022	0.022	31
	0.044	0.037	18.8
	0.062	0.048	14.5

The estimated data for 5.25% NaOCl are compared to actual data taken from Epstein (Epstein *et al.*, 1955) (constant hypochlorite concentration of 0.002 M). The data indicate that GB decomposition is promoted by increasing pH. Mildly acidic conditions discourage GB decomposition: at neutral pH, the half-life in 5.25% NaOCl is less than 5 seconds, while at pH 5 it increases to 64 minutes. This rate difference correlates with the mechanism proposed by Epstein (Epstein *et al.*, 1955), which involves GB reaction with OCl^-. The transition state, shown in Figure 2.4, is critically dependent on the anionic form, in which the polarizing effect and nucleophilic strength are maximized. As the pH decreases, the predominant species is HOCl. Although GB decomposition rate data are not available for pH < 5, the absence of OCl^- anions precludes hypochlorite-catalyzed hydrolysis. The decomposition in acid is likely dominated by simple hydrolysis. Hydrolysis rate data for GB in acidic solution are not available.

While data for the specific reactions of GA and GD with hypochlorite are not available, structural similarities between these agents and GB allow qualitative predictions relative to GB rates. In alkaline pH, both GA and GD are expected to react via hypochlorite-assisted nucleophilic displacement, as does GB, to produce rate enhancements relative to simple hydrolysis. Alkaline hydrolysis rates for both GA and GD are relatively high: GA hydrolysis at pH 9.5 and 25°C occurs with a pseudo-first-order rate constant of 0.02 min^{-1}, corresponding to a half-life of 35 minutes; GD decomposition is complete within 5 minutes in excess 5% sodium hydroxide (pH ~12.5) (Yurow and Davis, 1982). These data suggest that decomposition rates in hypochlorite, which—by analogy to GB—are expected to be higher than those for simple hydrolysis, should be very rapid for GD and moderately rapid for GA. Decomposition rates for GD in hypochlorite are expected to be comparable to those for GB due to the presence of the same leaving group (F^-), although GD rates will be somewhat slower because of increased hindrance to nucleophilic attack. Rates of GA decomposition in hypochlorite may be significantly slower than GB rates despite similar hindrance, primarily because GA decomposition involves a poorer leaving group (CN^- for GA versus F^- for GB). The cyanide ion formed from GA decomposition is readily decomposed in aqueous hypochlorite, alleviating any concern for potential cyanide toxicity (Yurow and Davis, 1982).

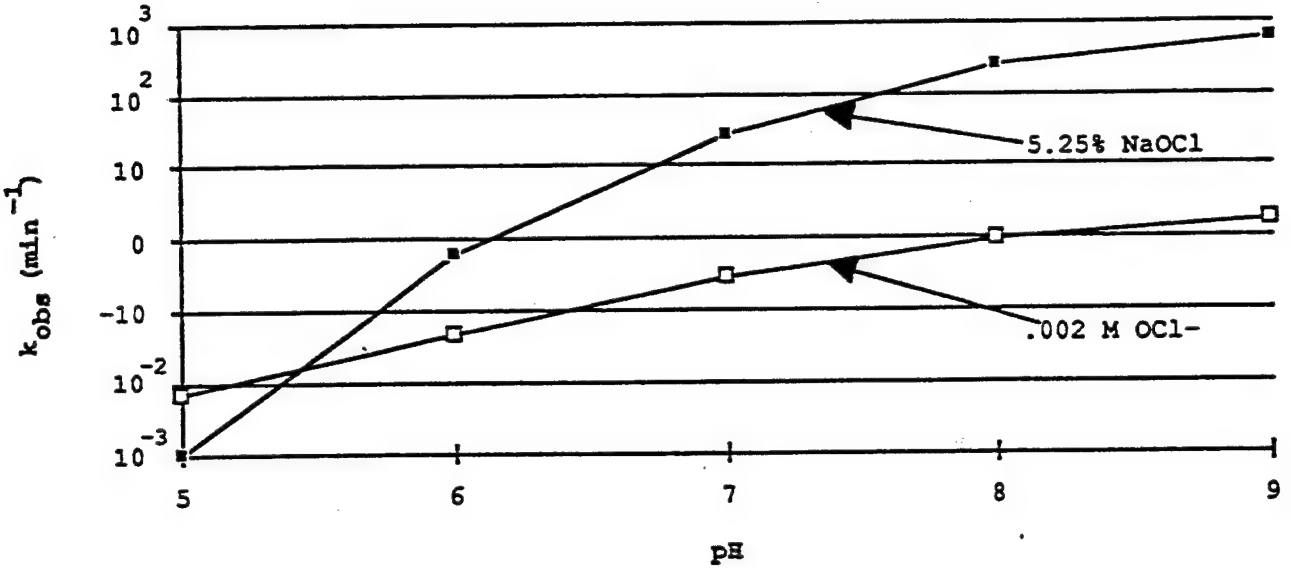


Figure 2.2. Rate of GB decomposition in aqueous NaOCl as a function of pH. Rates of GB decomposition are extrapolated to 5.25% NaOCl, the concentration of common household bleach. The strong pH dependence shown is principally due to the pH effect on [OCl^-] shown in Figure 2.1. A compounding pH effect on the hydrolysis of GB in aqueous NaOCl is illustrated by the extrapolation to solutions of a constant [OCl^-] of 0.002 M.

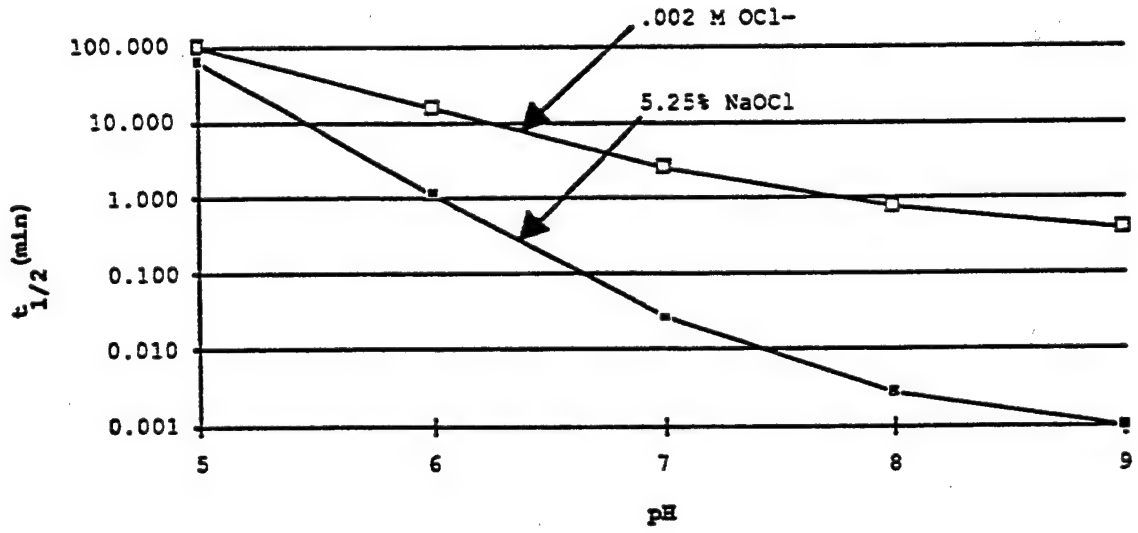


Figure 2.3. Half-life of GB in aqueous NaOCl as a function of pH. Half-lives of GB decomposition are extrapolated to 5.25% NaOCl, the concentration of common household bleach. The strong pH dependence shown is principally due to the pH effect on [OCl^-] shown in Figure 2.1. A compounding pH effect on the hydrolysis of GB in aqueous NaOCl is illustrated by the extrapolation to solutions of a constant [OCl^-] of 0.002 M.

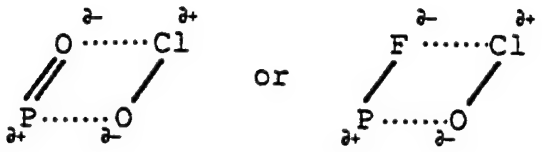


Figure 2.4. Proposed transition state for OCl^- reaction with G agents (Source: Epstein *et al.*, 1956).

2.2.3.2 Reaction of VX with Hypochlorite

VX reacts strongly with hypochlorite by a number of competing reactions. The products of VX decontamination are pH-dependent, with optimum conditions occurring in acidic solution. In the acidic pH range, the moderate solubility of VX is enhanced by protonation of its nitrogen atom. At low pH, hypochlorite solutions contain large amounts of aqueous Cl_2, and the reaction with VX occurs via acid chlorinolysis:

$$VX + 3\ Cl_2 + 4\ H_2O \rightarrow (CH_3)(C_2H_5O)\text{-}POOH + SO_3H\text{-}CH_2CH_2N(iso\text{-}C_3H_7)_2 + 6\ HCl$$

The half-life of VX at pH 4 and 25°C is 1.2 minutes (Yurow, 1981). This process has been used for the large-scale decontamination of VX (Yurow, 1981).

The pseudo-first-order rate constant for VX in 10% $Ca(OCl)_2$ at 25°C is 0.01 sec^{-1} (Yurow and Davis, 1982); thus the half-life of VX in this solution is approximately 1.2 minutes. The reaction of VX in strongly alkaline hypochlorite is nearly as rapid (VX half-life is 1.5 minutes at pH 10 and 25°C) (Yurow, 1981):

$$VX + 9\ OCl^- + 7\ OH^- = (CH_3)(C_2H_5O)\text{-}POO^- + HN(iso\text{-}C_3H_7)_2 + SO_4^{2-} + 2\ CO_3^{2-} + 9\ Cl^- + 5\ H_2O$$

Highly toxic products are formed by reaction of VX and hypochlorite in the pH range of pH 7-10 (Anderson, 1974). The toxic products are listed as diisopropylaminoethyl mercaptan, ethyl hydrogen methyl phosphonate, bis(ethyl methylphosphonic) anhydride, and bis-S-(2-diisopropylaminoethyl) methylphosphonodithioate (Anderson, 1974). To maintain reasonable rates while avoiding toxic products, the pH values for optimum VX decontamination are pH ≤ 4 and ≥ 11.

2.2.3.3 Reaction of HD with Hypochlorite

HD is readily decomposed by hypochlorite due to the presence of its oxidizable sulfur atom. It is well established that hypochlorite effectively decontaminates HD, and hypochlorite solutions have been recommended for a variety of practical situations involving HD decontamination (Yurow, 1981). HD is only sparingly soluble in aqueous hypochlorite. HD decontamination involves heterogeneous surface reactions. In acidic hypochlorite, HD decomposes according to the following reaction (Jody, *et al.*, 1983):

$$HD + 14\ HOCl \rightarrow H_2SO_4 + 16\ HCl + 4\ CO_2 + H_2O$$

In alkaline hypochlorite, the proposed reaction for HD decomposition is given by the same overall reaction as in acidic solutions, with the corresponding unprotonated products (Yurow, 1981). In alkaline solution, decrease in the hypochlorite concentration can lead to the formation of sulfoxide and/or sulfone products, which are somewhat toxic (Yurow, 1981).

Practical experience with large-scale decontamination of material indicates that HD is readily decomposed under both acidic and alkaline conditions. The reaction is expected to be complete within minutes, but no data are available to substantiate this expectation (Jody, *et al.*, 1983). This information gap prohibits the identification of optimal conditions for HD decontamination.

2.2.3.4 Reaction of Thickened Agents with Hypochlorite

While reactions of thickened agents with hypochlorite have not been characterized in the available literature, decontamination of the thickened agents is expected to be difficult. Thickened agents are insoluble in water. This insolubility prevents mixing with the decontamination solution, thereby hindering possible chemical reaction. While neat HD is also insoluble, HD can react with the decontamination solution at the surface interface and thus be decomposed. Thickened agents, however, are "buried" in a polymer lattice and are not accessible to solution at the phase interface. These solubility and sequestering effects suggest that chemical reaction between thickened agents and hypochlorite will be slow and will be controlled by

rates of agent desorption from the thickeners. Data on desorption rates are not available at present.

As a possible solution to difficulties anticipated for detoxification of thickened agent, testing of ultrasonic removal is suggested. Ultrasound is a well-established, readily available method which mechanically removes insolubles from surfaces by microscopic agitation. The agitation causes high local pressures and temperatures within the insoluble material. These serve to cavitate and implode surface contaminants, resulting in spalling and descaling of the contaminant from the surface. This method has been effective for solubilizing HD (Grotta *et al.*, 1983). The mechanical process underlying ultrasonic removal is anticipated to remove thickened agents effectively from skin surfaces and to force small particles of agent-thickener mixtures into the hypochlorite solution. The increased surface area of the small particles should enhance desorption of agent and overall reaction rates.

Actual rates of reaction between hypochlorite and ultrasonically removed agent-thickener particles are unknown, and comparisons to reaction rates of the neat agents are not possible. While ultrasonic removal is expected to enhance reaction rates of the thickened agents, the rates are still expected to be slow. Particles of ultrasonically removed HD can react immediately with decontamination solution at the interface between particle surface and solution. By comparison, in ultrasonically removed agent-thickener particles, agent is entangled in the thickener. Commonly used thickeners are expected to be inert to reaction with hypochlorite. In order for agents to encounter decontamination solution, it must desorb from the polymer. Rates of agent desorption are unknown. Nonetheless, the extra time required for desorption will slow reaction rates of thickened agents compared to neat agents.

The expected difficulties identified for thickened agents make it likely that decontamination rates for these agents will be slower than those for the unthickened agents. This possibility indicates that the effectiveness of a fielded decontamination system may depend critically on its ability to handle thickened agents. The lack of information regarding hypochlorite reaction with thickened agents must be remedied by testing and constitutes a serious information gap.

2.2.3.5 Summary of Kinetics Data

Consideration of the favorable conditions for agent decomposition reveal a limited range of adequate conditions for decontamination of all agents. Data on the decontamination of HD in alkaline hypochlorite are inadequate to assess the effects of pH change on efficiency. As such, optimal conditions for HD decomposition cannot be currently identified, but HD is known to be readily degraded by acidic and alkaline hypochlorite. Further, decontamination effectiveness with thickened agents is unknown. GB decomposition, and by analogy that for GA and GD, is optimized at pH > 7 and discouraged at lower pH values. The lack of rate data for GB in acidic solutions (pH < 5) is an information gap; nonetheless, acidic decomposition rates for GB are expected to be significantly lower than those in alkaline solution, as previously discussed. VX decomposition is favorable at pH < 7, unfavorable in the pH 7-10 range, and again favorable at pH ≥ 11. As a result, the sole conditions which are favorable for decomposition of both VX and G agents are alkaline solutions at pH ≥ 11. HD also decomposes readily at this pH, but at an unknown rate. Increasing the solubility of thickened agents and neat HD inherently increases reaction rates, but these rates are also unknown.

2.2.4 Temperature Dependence

The kinetics data for hypochlorite decontamination presented in Section 2.2.3 were generally obtained at 25°C. Field temperatures may be significantly lower. Lower temperatures tend to retard reaction rates. Assessment of a realistic worst-case scenario must consider the temperature dependence of agent-hypochlorite reaction rates. Data for GB obtained by Epstein (Epstein *et al.*, 1955; Epstein *et al.*, 1956) are presented in Table 2.3:

Table 2.3. Temperature dependence of hypochlorite decomposition of GB (concentrations not specified).

Temperature (°C)	k_{obs} (min^{-1})	$t_{1/2}$ (min)
3.5	0.0083	83
23.5	0.0307	23
34.0	0.0578	12

The data show that for every 10°C, the rate changes proportionally by a factor of two. This temperature dependence is typical for reactions of organic molecules. From data presented in Figure 2.3, the estimated half-life of 2×10^{-4} M GB in 5.25% NaOCl at 5°C and pH 10 is less than 1 second, while at pH 5 it is 256 minutes. The lack of dissolution rate data for HD, as well as dissolution rates and agent desorption rates for thickened agents,

prevents the estimation of these temperature-dependent half-lives.

While experimentally derived temperature-dependence data are not available for VX, it is expected that its behavior will also be typical. Assuming typical behavior for VX, the following expression for the termperature-dependent half-life can be derived from the half-life data of Yurow and Davis at 25°C (Yurow and Davis, 1982). (See also Table 2.4 and Figure 2.5.)

$$t_{1/2} = \frac{746}{[OCl^-]} \cdot 2^{(25-T)/10} \quad (seconds)$$

The estimated half-life of VX in alkaline hypochlorite at 5°C is 6.0 minutes at pH 10 and 4.8 minutes at pH 4 (hypochlorite and VX concentrations were not provided [Yurow, 1981]).

Table 2.4. Effect of hypochlorite concentration and temperature on the half-life (sec) of VX.*

Temp. (°C)	OCl^- Concentration (weight % NaOCl) 1%	3%	5%	7%	9%
0	4222	1407	844	603	469
5	2985	995	597	426	332
10	2111	704	422	302	235
15	1493	498	299	213	166
20	1055	352	211	151	117
25	746	249	149	107	83
30	528	176	106	75	59
35	373	124	75	53	41
40	264	88	53	38	29
45	187	62	37	27	21
50	132	44	26	19	15

*The values calculated for this table were used to generate Figures 2.5, 2.6, and 2.7.

Another important temperature-dependent parameter is agent solubility. GB and GA are miscible with water at expected field temperatures, and the temperature dependence of HD and GD solubility have not been found. The intuitive expectation that solubility increases with increasing temperature does not apply to VX. While VX is marginally soluble at 25°C (30 g/L), it becomes completely miscible at 9.4°C (Weyandt, 1991).

Mutual consideration of both rate and solubility dependence on temperature suggests a conflict in determining an optimal temperature for a fielded decontamination device. While higher temperatures promote decontamination by enhancing reaction rates, lower temperatures promote decontamination by increasing the solubility of certain marginally soluble agents, particularly VX.

Despite this apparent difficulty, the temperature dependence of agent solubilities is expected to be of low priority in determining optimal field temperatures. Under the expected field conditions, the quantity of poorly soluble agents such as VX is expected to be small enough to allow complete solubilization if the available volume of the decontamination bath is sufficient. Up to 1.3 kg of HD and 39 kg of VX can be dissolved at 25°C in a 1.3-m^3 decontamination vessel with proper mixing. These quantities are well beyond practically anticipated levels. Additional solubilization is offered by the incorporation of ultrasonic removal, particularly for thickened agents which are insoluble throughout the range of practically applicable temperatures. If implemented, ultrasound may provide sufficient solubilization regardless of temperature dependence effects. As such, the choice of temperature should be made on the basis of reaction rates rather than agent solubility. Additional concerns associated with a compromise between maximizing reaction rates

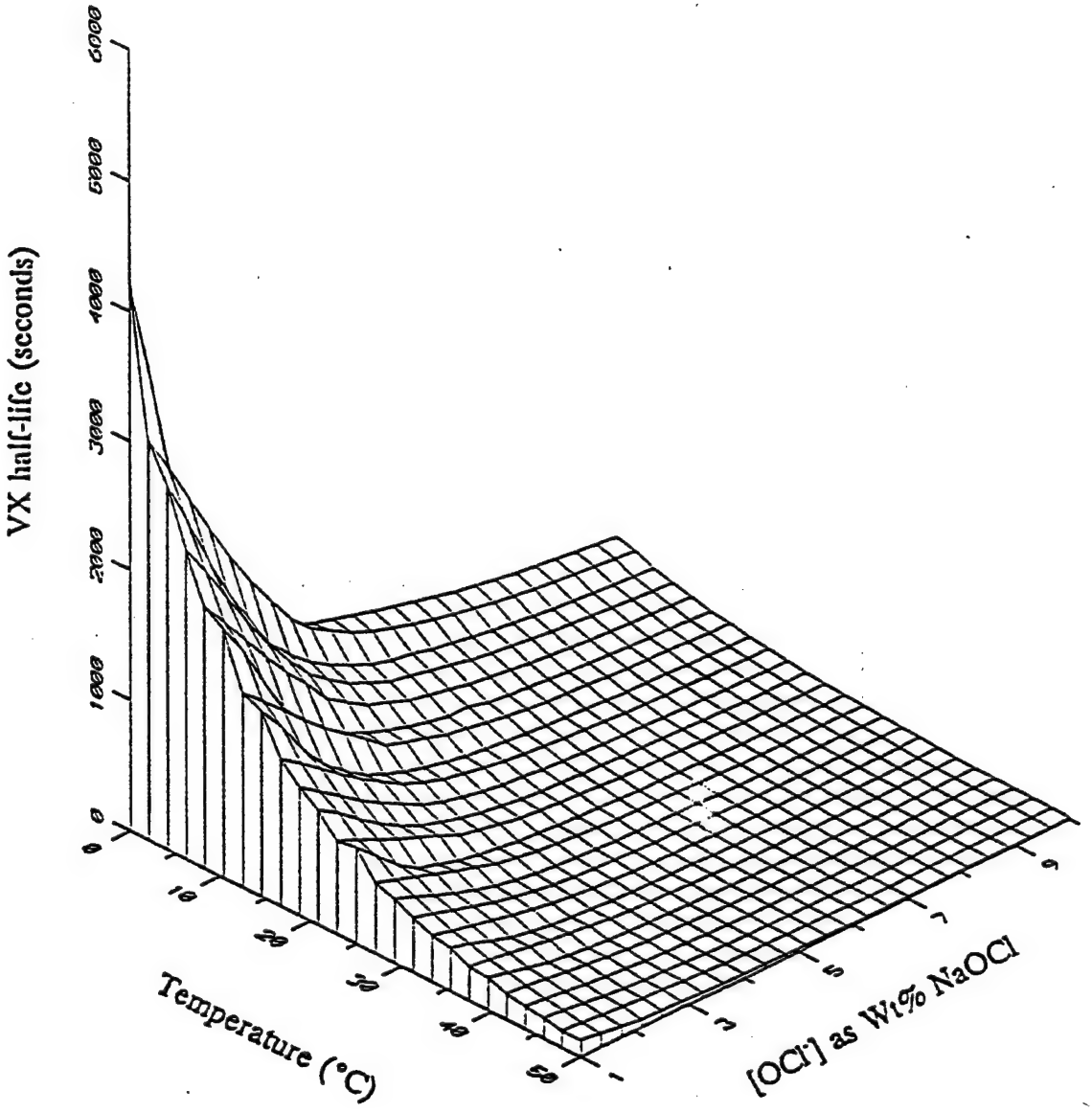


Figure 2.5. The half-life of VX as a function of OCl^- concentration and temperature. This three-dimensional graph shows that the half-life of VX is a monotonically decreasing function of both OCl^- and temperature. Thus concentrations of OCl^- below 5% exhibit long half-lives at low temperatures. Concentrations of OCl^- less than 3% produce VX half-lives between 1,000 and 6,000 seconds, whereas concentrations above 3% produce VX half-lives less than 1,000 seconds.

and minimizing cosmetic effects in determining the best temperature ultimately requires field validation

2.2.5 Effect of Hypochlorite Formulation

Aqueous solutions of hypochlorite can be derived from a variety of sources; the most common of these include NaOCl, calcium hypochlorite (HTH; $Ca(OCl)_2$), and HOCl solutions. The dependence of hypochlorite decontamination on the formulation of the hypochlorite solution has been determined by Epstein (Epstein *et al.*, 1956). Within experimental error, the rates in Table 2.5 are indistinguishable. The source of hypochlorite thus has no effect on the reaction rate. This finding is consistent with the proposed mechanism in which the OCl^- anion is involved in the transition state for the reaction, while various cations present are not.

Table 2.5. Effect of source of hypochlorite on rate of GB decomposition (concentrations not specified, Epstein *et al.*, 1956).

Source of Hypochlorite	Other Ions in Solution	Avg k (M^{-1} min^{-1})
HTH*	Ca^{2+}, Na^+, Cl^-	5.6
NaOCl	Na^+, Cl^-	6.0
HOCl	Na^+, NO_3^-	6.2

*Commercial hypochlorite formulation.

It has been suggested that different formulations are associated with enhanced decontamination efficiency, but these effects are expected to be negligible under field conditions. Under field conditions, however, the presence of buffers in decontamination solution may be required, and the buffers may dominate the ionic composition of the solution. Further, hypochlorite oxidation of organic material creates carbonate ion, and body fluids contain high concentrations of phosphate ions. Both calcium carbonate and calcium phosphate are insoluble and will precipitate from solutions containing high concentrations of Ca^{2+} ions. By contrast, all common sodium salts are soluble. The lack of rate enhancement demonstrated in Epstein's data (Epstein *et al.*, 1955; Epstein *et al.*, 1956), together with these considerations, suggests that sodium hypochlorite formulations are preferable.

2.2.6 Hydrolysis

The rates of simple agent hydrolysis are an important corollary to the issue of agent reaction kinetics. In the absence of catalysts and other agents which promote the decomposition of agents, hydrolysis is the primary chemical pathway for agent degradation. Examination of agent hydrolysis rates establishes a baseline for comparing alternative chemical decontamination methods and provides an indication of whether physiological fluids will chemically neutralize absorbed agents in the absence of enzymatic pathways. This analysis is important for assessing the potential persistence of absorbed agents in the body tissues and fluids of chemically contaminated remains.

All agents under consideration hydrolyze slowly at neutral pH and would be expected to persist in physiological fluids in the absence of alternative degradation pathways (i.e., enzymatic). Although HD hydrolyzes rapidly, the amount hydrolyzed is limited by its slow dissolution. The half-life of VX at neutral pH is 40 days at ambient temperature, while that for GB in dilute solutions is approximately 75 hours (Yurow, 1981). The rate of GD hydrolysis is expected to lie between those for VX and GB.

2.3 PRACTICAL IMPLICATIONS

2.3.1 General

The implementation of an effective decontamination procedure for treating chemically contaminated remains requires the consideration of the chemical kinetics discussed above as well as the careful examination of field-specific factors. Conditions for hypochlorite decontamination of all agents have been identified from a chemical perspective. The dependences of the observed rates on temperature, pH, and formulation have been outlined to the extent allowed by the available data. These findings provide the basis for estimating performance under conditions modeled to actual field conditions.

Within the limitations of the available data, it can be inferred that conditions for hypochlorite decontamination of the principal threat agents occur in alkaline solutions at pH 11. Rate data indicate that different hypochlorite sources have no effect on reaction rates and that sodium hypochlorite formulations are favorable for practical reasons. The following discussion will therefore be based on 5.25% NaOCl solutions. This

concentration corresponds approximately to 0.7 M NaOCl. Based on the acid dissociation constant of HOCl, the pH of fresh 5.25% NaOCl is approximately 10.6. The pH can be easily adjusted by adding small amounts of NaOH. Thus, 5.25% NaOCl is amenable to the preparation of the required solution.

A number of field-related concerns cannot adequately be estimated and require eventual resolution. There may be significant cosmetic effects associated with whole-body immersion in such a caustic solution. The field procedure should incorporate some mixing device to ensure complete mixing of agent with hypochlorite and thereby maximize the agent removal rates, particularly for thickened agents, HD, and decontamination at lower temperatures. In addition to reaction with dissolved agent, hypochlorite concentrations will be depleted by reaction with a variety of nonagent competitors such as skin, hair, body tissues, and physiological fluids. The presence of these interfering reactions, along with the agent-specific reactions, may have significant effects on the solution pH. Maintenance of proper solution pH is critical to ensure that toxic reaction products are not formed and may require the use of appropriate buffers. Resolution of these concerns ultimately requires validation.

2.3.2 Analysis of Selected Exposure Scenarios

Exposure scenarios can be examined to determine the feasibility of the field-scale decontamination process. The scenario presented here is based on the following assumptions:

- Agent is decontaminated using a hypochlorite solution derived from 5.25% NaOCl adjusted to pH 11.
- Total immersion in VX results in a 0.1-mm-thick residual agent film on skin having a surface area of 2 m^2 = 200 ml of agent to be decontaminated or ~200 g of VX.
- Exposure to VX delivered by munitions is estimated to be a maximum total of 4 g.
- VX decontamination represents the worst case in terms of reaction kinetics. VX has a longer half-life than GB under these conditions (the reaction rates of HD, GD, THD, and TGD are not known for these conditions, but the nonthickened agents are expected to have shorter half-lives than VX).
- A field temperature of 5°C is considered to be worst case (lower temperature will require thawing). Agent half-lives can be extrapolated from available data assuming a twofold proportional rate change for a 10°C temperature change.
- The solution volume of the decontamination vessel is 1.3 m^3.
- The experimentally determined first-order rate constant for VX decomposition in 10% $Ca(OCl)_2$ at 25°C is 0.01 sec^{-1} (Yurow and Davis, 1982). From this data, the half-life as a function of temperature and hypochlorite concentration can be derived as:

$$t_{1/2} = \frac{746}{[OCl^-]} \cdot 2^{(25-T)/10} \quad (seconds)$$

- Optimal processing time for reducing VX concentrations to nontoxic levels is approximately 15 minutes to avoid extensive cosmetic effects. The standard for acceptable levels is taken as the drinking water standard: 20 ppb (ERDEC standard). This translates to maximum VX half-life of approximately 145 seconds.

Three exposure scenarios are examined; they address contamination levels of 10 grams, 100 grams, and 200 grams of VX. Ten-gram exposures approximate the contamination expected to be delivered to remains by munitions, while 100- and 200-gram exposures represent levels resulting from whole-body immersion. Each scenario addresses the factors that are involved in achieving decontamination to the 20-ppb level. These factors include temperature, time, and OCl^- concentration. Table 2.6 shows the relationship of temperature and time, while Figures 2.6 and 2.7 graphically represent the VX half-life as a function of OCl^- concentration and temperature.

Table 2.6. Effect of temperature (°C) on time (min) to decontaminate VX to 20 ppb.

	10 g	100 g	200 g
5	90	120	130
25	22	29	31
30	15	–	–
34	–	15	–
35	–	–	15

In the first exposure scenario, dissolution of 10 grams of VX produces an initial agent concentration of approximately 8 ppm in 5.25% NaOCl decontamination solution. Using the worst-case field temperature (5°C), the half-life of VX in decontamination solution is approximately 10 minutes. Nine half-lives are required to reduce an initial VX concentration of 8 ppm to 20 ppb. At this temperature (5°C), nine half-lives of VX in 5.25%

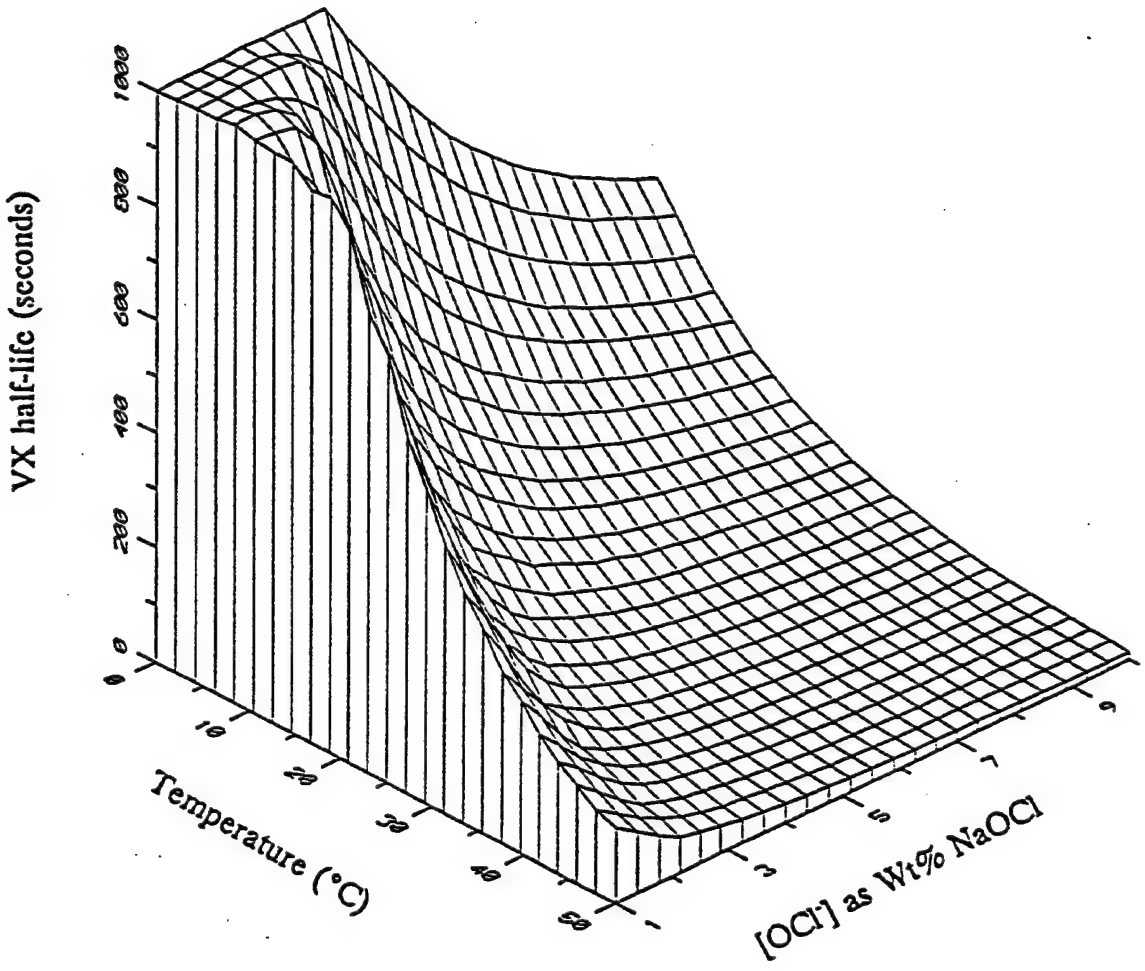


Figure 2.6. A view of the VX half-life up to 1000 seconds plotted as a function of OCl^- concentration and temperature in a contour graph.

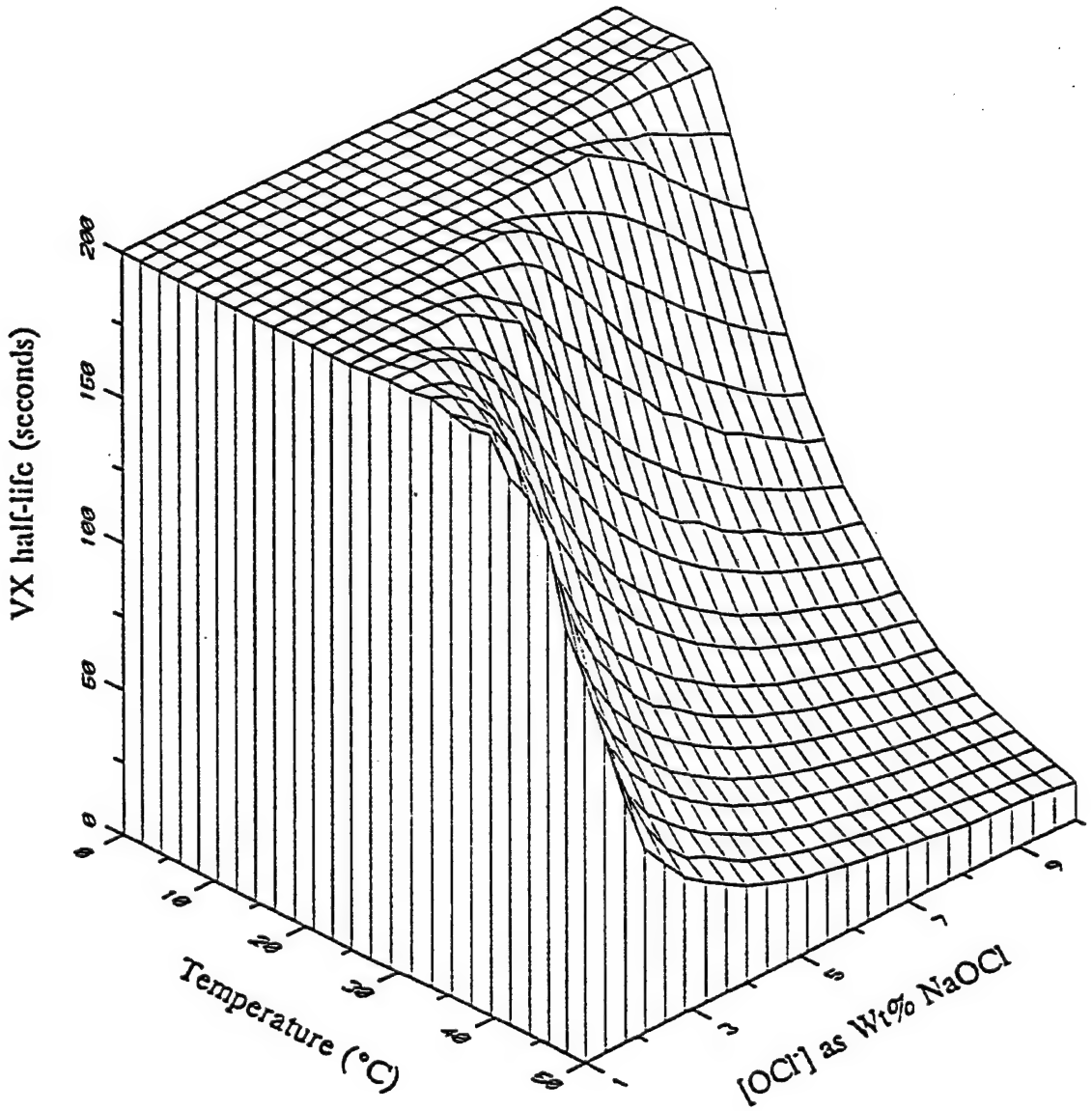


Figure 2.7. VX half-lives up to 200 seconds plotted as a function of temperature and OCl^- concentration in a contour graph.

NaOCl are approximately 90 minutes. At 25°C, the reaction rate is increased fourfold, and the corresponding half-life of VX in 5.25% NaOCl is approximately 2.4 minutes, so the 20-ppb level is achieved in 22 minutes. The necessary temperature needed to achieve the 20-ppb level, using 15 minutes as the targeted processing time, is 30°C. At 5°C, the level of decontamination achieved is 2 ppm in 20 minutes, compared to 125 ppb in 15 minutes at 25°C.

In the second scenario, dissolution of 100 grams of VX produces an initial agent concentration of approximately 76 ppm in 5.25% NaOCl decontamination solution. At 5°C, the time required to reach the 20-ppb level is 120 minutes compared to 29 minutes at 25°C. The temperature needed to achieve the 20-ppb level, within 15 minutes, is 34°C. At 5°C, the level of decontamination achieved is 19 ppm in 20 minutes, compared to 1 ppm in 15 minutes at 25°C.

In the worst-case scenario (total immersion), dissolution of 200 grams of VX produces an initial agent concentration of approximately 154 ppm in 5.25% NaOCl decontamination solution. At 5°C, the time required to reach the 20 ppb level is 130 minutes compared to 31 minutes at 25°C. The temperature needed to reduce the VX concentration to 20 ppb, within 15 minutes, is 35°C. At 5°C, the level of decontamination achieved is 39 ppm in 20 minutes, compared to 2.4 ppm in 15 minutes at 25°C.

Figures 2.6 and 2.7 reinforce the above calculated temperature and concentration effects on VX half-life. The plots indicate that OCl^- concentrations less than 3% give VX half-lives in excess of 1000 seconds. For NaOCl concentrations between 3% and 5%, there are steep gradients and the half-life is easily affected by temperature fluctuations. At NaOCl concentrations greater than 5%, the gradient is attenuated and temperature fluctuations have a less pronounced effect on the half-life of VX.

In an effort to identify a processing time within practical limits, Figure 2.7 provides a closer look at VX half-lives up to 200 seconds plotted as a function of temperature and OCl^- concentration. This figure indicates that to meet this criterion, the OCl^- concentration must be at a minimum of 5% NaOCl and the temperature range should be between 15°C and 25°C.

The possible use of more dilute hypochlorite solution to minimize cosmetic effects is not advisable on the basis of these rate data. Kinetic studies demonstrate that reactions with G agents are first order with respect to hypochlorite concentration. While VX decontamination proceeds by a different mechanism, estimates of the rate effects associated with dilute hypochlorite can be made by assuming first-order kinetics. These estimates indicate that reducing the hypochlorite concentration from 5.25% to 0.5% would result in a tenfold decrease in reaction rates and an associated tenfold increase in agent half-life. At 25°C and pH 11, the increase in the half-life would result in residence times on the order of several hours to ensure complete detoxification. This increase in required decontamination time is impractical.

Table 2.7 was constructed to compare the effectiveness of decontamination of the maximum amount of VX to which remains would be exposed by the three exposure scenarios.

Table 2.7. Effect of temperature and hypochlorite concentration on time (min) to degradation of VX to 20-ppb level.

Total Agent to Decontaminate (g)	10†		100		200††	
Temperature of decontamination solution(°C)	5	25	5	25	5	25
Hypochlorite Concentration						
5.25%	90	22	120	29	130	31
0.5%	900	216	1200	288	1300	312

†Estimated total agent remains would be exposed to if delivered by munitions.
††Estimated total agent remains would be exposed to if immersed in puddle.
Calculations were done using values found in Sections 3 and/or 4.

The estimated maximum amount of VX to be decontaminated following total immersion is calculated to be 200 g. The estimated maximum amount of VX to be decontaminated following exposure of the remains to agent delivered by munitions is 10 g. If the concentration of the decontamination solution is reduced to 0.5%, it would take 216 minutes to decontaminate 10 g of VX in a 1.3-m^3 tank at 25°C.

The limitations imposed by the volume of the decontamination tank, the time for the decontamination soak of the remains, and the calculated amount of agent to be decontaminated dictate that 0.5% hypochlorite would not be sufficient for use as a decontamination solution. The lower concentration would be adequate for decontamination of personnel, wounds, and equipment with less agent contamination.

The maximum pH change created by agent decomposition alone can be estimated to determine the severity of pH control problems arising from the agent decontamination process. Examination of the stoichiometry of each agent reaction with hypochlorite indicates that the greatest pH change accompanies the degradation of HD: 18 moles of hydrogen ion are produced for each mole of HD decomposed. For 100 ml of HD (0.8 mole), 14.4 moles of hydrogen ion are generated. The pH of unbuffered 5.25% NaOCl is reduced to 2.1 by the decomposition of this amount of HD. This large pH change depletes active OCl^- anions, defeats the maintenance of appropriate solution pH, and thus undermines G agent decontamination.

In addition to the pH change caused by agent decomposition, the presence of body fluids (which tend to be buffered around pH 7) will influence the pH of the decontamination solution. Determination of the extent of this effect requires testing. Whether the pH change is governed by agent degradation or by the presence of body fluids, the decontamination solution will need to be buffered. Potentially useful buffers must maintain the solution pH at values near 11. Possible choices include mixtures of sodium hydroxide and sodium bicarbonate or disodium hydrogen phosphate (Weast, 1989). The final choice of buffer requires validation by field testing.

2.4 CONCLUSIONS

The implementation of an effective field decontamination procedure for the decontamination of chemically contaminated remains requires the consideration of the underlying chemical processes. Examination of the reaction rates of hypochlorite-agent reactions suggests that hypochlorite solutions are effective surface decontaminants for the principal threat agents, although efficacy against thickened agents is not established. Optimal conditions for decomposition of all agents do not exist due to rate differences and generation of toxic products as a function of pH. Mutually adequate conditions for pH and temperature have been identified. A solution pH of 11 has been identified on the basis of reasonable reaction rates with both VX and GB and avoidance of toxic products from VX degradation. Temperatures near 25°C allow sufficient reaction times to provide detoxification of residual VX (the rate-limiting agent based on available data) and are optimal for reaction times of $\leq$ 15 minutes. Lower temperatures retard reaction rates sufficiently to require longer practical decontamination times.

Temperature and pH control are critical to successful decontamination, and the design of actual field systems must incorporate features, such as appropriate heating and buffering systems, which control these parameters. The pH variation of the decontamination solution will be influenced by the presence of body fluids from the contaminated remains. The magnitude of this influence and the details of the pH variation during actual decontamination require test validation.

Additionally, the low solubilities of HD, VX, GD, and the thickened agents suggest that mixing systems should be incorporated. Ultrasound offers the potential for maximizing reaction rates by increasing solubility without creating additional cosmetic effects. Ultrasound is a readily available, proven technology. The possible application of ultrasound should be considered and tested.

The formulation of the hypochlorite solution does not significantly affect reaction rates, but practical considerations indicate that sodium hypochlorite solutions are preferable. Calcium formulations result in the formation of precipitates as decontamination proceeds and are not amenable to the preparation of necessary buffer solutions. Both sodium and calcium formulations are readily available. Thus, sodium formulations provide efficient decontamination solutions without unwanted practical drawbacks.

The concentration of hypochlorite solution significantly affects the rate of decontamination. For the quantities of agent to be decontaminated in the specified worst-case scenario, decomposition rates were inadequate for 0.5% hypochlorite solutions, whereas 5.25% solutions provided adequate decontamination times. The efficacy of decontamination is demonstrated in the ability of 5.25% NaOCl at 25°C to reduce an initial VX concentration of 8 ppm to 20 ppb in 22 minutes, whereas a 0.5% solution requires over 3 hours to reduce 8 ppm VX to the 20-ppb level.

Several important implications are suggested by results of the model scenarios. Exposures representative of munition-delivered agent (i.e., 10 g) can be decontaminated to drinking water levels by 5.25% hypochlorite at 25°C in 22 minutes. While this exceeds the selected optimal time of 15 minutes, this residence time was arbitrarily selected, and it is unclear whether the additional 7 minutes residence time will significantly exacerbate cosmetic effects. Nonetheless, a single 15-minute residence in 5.25% NaOCl at 25°C reduces the VX concentration to 125 ppb. While this concentration exceeds the drinking water standard, it is perhaps sufficiently low to be considered essentially detoxified. Though the details of these issues ultimately require field testing for resolution, it appears that 5.25% NaOCl solutions at 25°C are capable of achieving the required levels of decontamination within practical residence times near 15 minutes.

In order to achieve complete detoxification of exposures to 100-200 g, two successive 15-minute immersions are required. Given the remote likelihood of these contamination levels, the additional cosmetic effects associated with the repeated residence may not be of concern. In the event that excessive cosmetic effects result from the repeated decontaminations, alternative decontamination methods may be more practical. The extent of additional cosmetic effects associated with successive immersions, as well as the feasibility and desirability of alternative decontamination methods, requires field validation. Nonetheless, it appears that two successive immersions will provide adequate decontamination at these high levels of contamination.

The wastewater generated during decontamination procedures should be handled as contaminated waste. The by-products of agent decontamination, while less toxic than pure agent, are nonetheless sufficiently toxic to treat cautiously. Fluoride salts and low concentrations of mercaptans, sulfoxides, sulfones, and organophosphates are all by-products of agent decomposition and are subject to prudent waste disposal.

3. POTENTIAL HAZARD TO HANDLERS OF DECONTAMINATED REMAINS

3.1 INTRODUCTION

Even if the surface decontamination of chemically contaminated remains is completely successful, active agent not accessible to the decontaminant may still be present in the body. This agent could pose a hazard to handlers, such as morticians and pathologists, who would come into intimate contact with the remains during embalming and autopsy procedures. Residual contamination is favored by prolonged contact of the remains with agent prior to decontamination--a period which could exceed 24 hours. The objective of the following analysis is to obtain an estimate of the potential transfer hazard of toxic agent to handlers of the decontaminated remains.

The overall strategy employed for the assessment of the potential transfer hazard is as follows:

(1) Develop battlefield scenarios for worst-case exposure to chemical agents. For these scenarios, obtain estimates of the maximum levels of chemical contamination that can be sustained by the casualty.

(2) The threat agents of concern are mustard gas (HD), tabun (GA), sarin (GB), soman (GD), thickened soman (TGD), VX, and thickened VX (TVX).

(3) Obtain estimates of the maximum quantity of active agent that could be sequestered in the body of the casualty and would not be susceptible to surface decontamination.

(4) Ascertain the fate of the agent in the body by analyzing the efficacies of various modes of *in vivo* detoxification and/or agent disposition. These mechanisms are: reaction with specific and nonspecific targets, spontaneous and/or enzymatic hydrolysis, metabolic conversion, excretion, and attenuation of toxicity by dilution. Of particular interest is the possible existence of agent depots or reservoirs that could store large quantities of active agent and could subsequently pose a hazard to handlers.

(5) Estimate the potential hazard of residual agent to handlers of the decontaminated remains. Identify such potential hazards as vapor and/or contact hazards.

3.2 POTENTIAL LEVELS OF BATTLEFIELD CONTAMINATION: WORST-CASE SCENARIOS

To ascertain potential levels of contamination of personnel exposed to munition-delivered chemical threat agents, it is necessary to consider the level of such agents that might be encountered on the chemical battlefield. These factors will influence contamination levels:

(1) The physicochemical characteristics of the agent under consideration.

(2) The type of munition used and the method of agent dispersal (the fireplan).

(3) Environmental factors such as temperature, humidity, and wind speed.

(4) The proximity of personnel to peak concentrations of the agent.

(5) The duration of exposure.

(6) The protective equipment and/or clothing worn.

(7) The extent of pretreatment or treatment received.

(8) The timing of exposure with respect to the time of death.

(9) The rate of agent penetration or absorption.

(10) The presence of skin abrasions or open wounds that might enhance agent entry.

Another scenario for potential massive exposure is the total immersion of the remains in a puddle of agent.

The main objective of this analysis is to protect all handlers of the decontaminated remains from any possible harm from exposure to active agent that may persist in the casualty. It is thus necessary to consider the battlefield exposure scenarios from the viewpoint of the handler rather than from that of the casualty exposed to agent.

As a starting point in evaluating the potential for agent exposure by handlers of the remains, it is assumed that the following conditions prevail:

(1) An unclothed casualty is exposed to peak levels of liquid agent and to maximum levels of vaporized (or aerosolized) agent that may be encountered on the battlefield. (It is expected, however, that liquid agent would pose a greater contamination hazard since maximum vapor levels will be present for only short periods.)

(2) The area of exposed bare skin is 2 m^2. Protective clothing is absent.

(3) Hazardous levels of active agent inaccessible to the surface decontaminant have accumulated and are confined to tissue depots (e.g., in the epidermis or body fluids).

(4) The casualty either was dead at the time of exposure or succumbed shortly after exposure to

supralethal levels of agent (especially of nerve gases). A dead casualty, who lacks both respiratory and circulatory functions, is more likely to accumulate hazardous levels of agent in the epidermis than is a living casualty, in whom the agent would be rapidly dispersed and diluted in the body.

(5) Skin absorption of agent is likely to result in agent accumulation in the epidermis. The extent of cutaneous absorption of an agent depends on its rate of penetration relative to its rate of evaporation. It is anticipated that VX, the low-volatility agent (which has the highest sustained percutaneous penetration of the agents considered here), would pose a significantly greater contamination hazard to handlers of the decontaminated remains than would the other agents. In turn, intermediate-volatility agents such as GA, GD, and HD, would be more hazardous than GB, the high-volatility agent. Also, thickened agents, which have lower evaporation rates (and, therefore, higher contamination potentials), would be expected to be more hazardous to handlers than the corresponding neat agents.

(6) The lower the rate of detoxification of agent in the body, the higher the potential risk to handlers of the decontaminated remains. The threat agent with the lowest *in vivo* detoxification rate is VX. The only probable means of VX detoxification in humans appears to be spontaneous hydrolysis, which proceeds at a slow rate (see Section 3.3).

(8) The greatest hazard to handlers could ensue if toxic levels of a low-volatility agent were dissolved in body fluids and would fail to be detected by the Chemical Agent Monitor (CAM) or other agent vapor-detection devices. Because of its low vapor pressure, VX is such an agent. VX contamination, therefore, could pose a significant contact hazard to morticians and others involved in handling the remains.

3.2.1 Strategy for Estimating Active Agent Levels in Contaminated Remains

Two approaches were considered for obtaining estimates of residual agent levels which may be present in decontaminated remains. One strategy involves the use of human percutaneous penetration rates of chemical agents to calculate the quantity of agent entering the body prior to decontamination. A second strategy was explored wherein estimates of human percutaneous toxicity are used and the levels of contamination of the remains are expressed in toxicity units (LD_{50}s). The use of toxicity units for estimating the contamination levels, which is the approach employed in this report, was deemed superior to the agent penetration approach for the following reasons:

(1) By focusing on toxicity rather than on initial levels of percutaneously absorbed chemicals, it more accurately reflects levels of active agent present in the body.

(2) By focusing on toxicity, it automatically makes allowances for the early hydrolysis, detoxification, and dispersion of the agent in the body (continuing detoxification and dispersion of agent would be expected, however, before the remains are handled by morticians or pathologists).

(3) It makes no questionable assumptions regarding the duration of exposure of the remains to the agent, which may vary greatly (more than 24 hours could elapse prior to decontamination). Duration of exposure is a necessary parameter for calculating agent absorption by the penetration rate method.

(4) It makes no questionable assumptions regarding the extent of the skin area covered with contaminating agent, which can vary widely in different exposure scenarios. The area of skin covered is a necessary parameter for calculating agent absorption by the penetration-rate method (only in the case of whole-body immersion in agent is the area coverage reasonably well approximated).

(5) It allows for ready comparison of active agent levels following absorption by different routes of administration.

(6) It allows ready comparison of toxic hazards associated with contamination by different agents.

(7) It permits an evaluation of the maximum initial hazard to handlers (subject to subsequent further detoxification in the body) due to exposure to contaminated remains. It should be emphasized that the use of toxicity units for estimating the contamination of remains is not meant to imply anything about toxic mechanisms or effects expected in the casualty. Rather, the data are thought to be a reflection of how much active agent could have entered the remains. The eventual hazard to handlers will be determined by the fate of the agent in the body prior to handling (see Sections 3.3 and 3.4).

Agent in both the liquid and vapor (or aerosol) forms can contribute to the hazard of the decontaminated remains. Thus it is necessary to ascertain how much of each form could penetrate into the body tissues and fluids and thereby remain inaccessible to surface decontamination. This analysis makes the following additional assumptions: (1) The casualty was dead at the time of exposure or died shortly afterward. These conditions would prevent the dispersion and subsequent dilution of the agent by the

respiratory and circulatory systems. Although this assumption is realistic for exposure to supratoxic levels of nerve agents, it may not hold for large exposures to HD, which produce lethal effects after a delay of several hours.

(2) The cessation of breathing will essentially eliminate any accumulation of agent by inhalation. It is therefore assumed that the hazard of agent in decontaminated remains derives primarily from penetration of both liquid droplets and agent vapors through the skin of the casualty. Vapor contamination of skin will be negligible, however, because peak vapor levels will be maintained for only a short time.

(3) The cessation of blood flow in a dead casualty and the slow rate of diffusion limit the entry of cutaneously absorbed agent into body fluids. Thus high agent levels could accumulate in the epidermis and surrounding tissues. Indeed, sequestration of VX, inaccessible to surface decontamination by bleach, was demonstrated in the human epidermis when exposure of volunteers was performed at cool temperatures (Cummings and Craig, 1965; Craig, *et al.*, 1977); such accumulation would be likely to occur in dead casualties. (It should be noted, however, that upon entry of the volunteer into a warm environment, which increases dermal circulation, the sequestered VX entered the bloodstream and inhibited blood cholinesterase activity [Craig, *et al.*, 1977].) (See Section 3.3)

(4) All of the agent remaining on the skin or wound surfaces at the time of decontamination of the remains was successfully removed by the decontaminant.

3.2.2 Battlefield Munitions

McNally *et al.* recently published an extensive analysis of the worldwide chemical and biological threat to U.S. air bases (McNally *et al.*, 1992). The modeling of chemical attacks on U.S. air bases (see Appendix B of McNally *et al.*, 1992; [Appendix A of this document]) provides relevant information on battlefield contamination potentials of a variety of chemical weapon systems. These battlefield scenarios can be used to help estimate the potential for exposure of personnel to various levels of chemical agent. Threat systems that were considered in this analysis included attacks by short-range munitions (mortar attacks) or moderate-range systems (aircraft bombing attacks and ballistic missile attacks). Agent fills studied were sarin (GB), mustard gas (HD), soman (GD), thickened soman (TGD), VX, and thickened VX (TVX). Although not comprehensive, the threat systems presented represent a significant array of fireplans for known fielded or developed systems. However, nonexploding types of munitions, such as aerosol generators and spray tanks, were not analyzed. Chemical agents dispersed by sprays from low-flying aircraft could increase agent deposition densities by up to tenfold beyond those produced by bombs, artillery, or tactical ballistic missiles (McNally, personal communication). Accordingly, estimates of potential peak contamination levels of remains will have to be revised upward as new data become available.

McNally *et al.* pointed out that as important as the fireplan and munition characteristics are, weather conditions can greatly influence the dispersion and transport of the agent following release from the munition (McNally *et al.*, 1992). Three meteorological conditions were presented for each attack: (1) low temperature (4°C) and low wind speed (1.5 m/sec), typical conditions for night release of agent during winter in temperate climates; (2) moderate temperature (25°C) and moderate wind speed (3 m/sec), typical dawn and dusk conditions during spring or fall in temperate climates; and (3) extreme conditions (49°C and wind speed of 6 m/sec), typical conditions during summer in areas such as Southwest Asia. Condition (1) provides the highest potential level of contamination with chemical agents because of their greater persistence. Condition (1) exposures would potentially pose the greatest hazard to handlers of the remains. (See McNally *et al.*, 1992 [Appendix B of this document] for the time required for 90% evaporation of various chemical agents as a function of temperature and wind speed.[1])

Deposition area coverage charts for the low-temperature, low-wind speed conditions were used to show total liquid deposition of each agent (in mg/m^2) caused by an attack with different munition systems (McNally *et al.*, 1992). This information was then used to estimate the peak levels of HD, GB, GD, TGD, VX, and TVX that can be expected on a target of 2 m^2 which, for present purposes, represents an unclothed, dead casualty. Table 3.1 shows estimates of peak liquid exposures to casualties. As mentioned previously, higher peak exposures to liquid agent are possible with other munitions or other attack modes, e.g., agent sprays from low-flying aircraft.

[1]Although the data in McNally *et al.*, 1992, compare the evaporation of neat and thickened agents from short grass, analogous information can be calculated for agent evaporation from other surfaces (e.g., skin). The higher persistence of 90% of the agent in the thickened form compared to the neat agent is due to the larger droplet size of the thickened agents and not to any basic difference in evaporation rates. The remaining 10% of thickened agents will persist even longer due to the formation of gels, which have lower evaporation rates.

Table 3.1. Potential battlefield contamination of casualties by chemical agents released from explosive munitions (Sources: McNally *et al.*, 1992; Anderson, 1974; Clement, 1984).

Agent	Munition	Liquid Exposure (Peak Levels[a]) (g/man)[c]	$(LD_{50})^{\dagger}$	Vapor Exposure (Maximum Dosage[b]) ($mg \cdot min/m^3$)	$(LCt_{50})^{\dagger\dagger}$
HD	Mortar attack; 36 120-mm rounds; 2 kg HD/round	20	6.7[d]	ca. 1,400	ca. 0.9(inhalation)[e] ca. 0.14 (skin)[e]
	Aircraft bombing attack; 96 100-kg ground-burst bombs; 30 kg HD/bomb	20-200	6.7-67[d]	ca. 10,000	ca. 6.7(inhalation)[e] ca. 1.0 (skin)[e]
GB	Aircraft bombing attack; 32 250-kg ground-burst bombs; 50 kg GB/bomb	20-200	11.8-118[f]	10,000	143 (inhalation)[f] 0.8 (skin)
	Tactical ballistic missile attack; 100 submunitions, each containing 2 kg GD	2-20	1.2-11.8[f]	1,000	14 (inhalation)[f] 0.08 (skin)
	Tactical ballistic missile warhead filled with GB[g]	200	118[f]	10,000	143 (inhalation)[f] 0.8 (skin)
GD	Tactical ballistic missile attack; 100 submunitions, each containing 2 kg GD	2-20	5.7-57[h]	ca. 600	ca. 8.6 (inhalation)[h] (skin)[h]
TGD	Aircraft bombing attack; 32 250-kg airburst bombs; 50 kg TGD/bomb; 2500-μm droplets	20-200	57-570[i]	10,000	143 (inhalation)[i] (skin)[h]
	Bulk release attack by tactical missile warhead; 500 kg GD	20	57[i]	1,000	14.3 (inhalation)[i] (skin)[h]

(continued)

$^{\dagger}LD_{50}$ = Number of percutaneous LD_{50}s = g/casualty/g/human LD_{50}.

$^{\dagger\dagger}LCt_{50}$ = Number of inhaled or percutaneous LCt_{50}s = $mg \cdot min/m^3$/vapor inhalation or percutaneous LCt_{50} for humans.

Table 3.1. Potential battlefield contamination of casualties by chemical agents released from explosive munitions (continued).

Agent	Munition	Liquid Exposure (Peak Levels[a]) (g/man)[c]	$(LD_{50})^{\dagger}$	Vapor Exposure (Maximum Dosage[b]) (mg·min/m³)	$(LCt_{50})^{\dagger\dagger}$
VX	Tactical ballistic missile attack; 100 submunitions, each containing 2 kg VX	2-4	200-400[j]	k	
TVX	Bulk release of VX from tactical ballistic missile; droplet diameter slightly above 300 microns	6	960[l]	k	

[a]From area coverage chart; 4°C, 1.5-m/sec wind speed (i.e., highest persistence) (McNally *et al.*, 1992).
[b]From dosage area coverage charts. Dosage is the time-integrated vapor concentration history and is used to characterize peak vapor dosage level expected on the target. Maximum vapor dosage is sustained at low temperatures and wind speed (McNally *et al.*, 1992).
[c]Assumes unclothed man with 2 m^2 of exposed skin.
[d]According to Papirmeister *et al.*, 1991, LD_{50} of liquid HD ranges from 3000-7000 mg/man; the 3000-mg value was used to calculate the number of percutaneous LD_{50}s for the worst-case scenarios.
[e]According to Tomlinson and Samuel, 1980, the LCt_{50} for HD vapor by inhalation is 1500 mg·min/m^3; the LCt_{50} for HD vapor by skin absorption is 10,000 mg·min/m^3.
[f]According to Anderson, 1974, the LD_{50} of liquid GB on bare skin is 1.7 g/man; the LCt_{50} of GB vapor by inhalation is 70 mg·min/m^3 at respiratory minute volume of 10 L/min; the LCt_{50} of GB vapor on bare skin is 12,000 mg·min/m^3.
[g]This type of fuse-operated explosive results in large contamination densities and large droplet sizes which are more persistent than those produced by other explosive devices (McNally *et al.*, 1992).
[h]The skin LCt_{50} for GD vapor is not known (Tomlinson and Samuel, 1980). The total hazard dose does not include any percutaneously absorbed GD vapor. The percutaneous toxicity of GD liquid is estimated at 0.35 g/man on bare skin. The LCt_{50} of inhaled GD vapor is estimated to be 70 mg·min/m^3 for a man breathing 15 L/min.
[i]Significant data voids exist regarding the percutaneous effectiveness of both increased temperature and thickened GD (Tomlinson and Samuel, 1980). The estimated toxicities for TGD employed the toxicity data for GD (see footnote h) and thus may be underestimated.
[j]The estimated percutaneous LD_{50} of VX liquid is 10 mg/man (Anderson, 1974). The estimated LCt_{50} by inhalation of VX aerosol is 30 mg·min/m^3 at a breathing rate of 15 L/min. The estimated LCt_{50} by percutaneous absorption of VX aerosol on bare skin is 6-360 mg·min/m^3 at wind speeds of 15-1 mph, respectively (Anderson, 1974).
[k]Volatility too low to pose vapor hazard.
[l]TVX is 1.6 times more toxic than neat VX (Tomlinson and Samuel, 1980).

Dosage[2] area coverage charts, which shows vapor dosages at various times following an attack, were used to characterize the peak vapor dosage levels for agents HD, GB, GD, TGD, VX, and TVX to be expected on a target–in this case, an unclothed casualty with 2 m^2 of skin surface. Table 3.1 shows that maximum vapor dosages generated by attacks with different munition systems were estimated to range from 1,400-10,000 mg·min/m^3 for HD to 600 mg·min/m^3 for GD, and were estimated to be unmeasurably low for VX or TVX (McNally *et al.*, 1992). Vapors of high-volatility agents such as GB can attain high dosage levels very rapidly. Vapors of intermediate-volatility agents such as GA, GD, and HD tend to take hours or days after an attack to generate dosage levels. The time required to achieve optimal dosage levels thus is critically dependent on the volatility of the agent. VX, with a very low vapor pressure, does not pose a vapor contamination hazard with the munitions considered here (McNally *et al.*, 1992). Nonexplosive release of VX by aerosol generators or spray tanks could, however, significantly increase VX vapor levels for short periods. Vapor dosages to casualties for all agents released by spray devices also would be expected to be higher than those estimated for explosive-release-type munitions. In no case, however, would the remains be exposed to peak vapor concentrations for prolonged periods, and percutaneous vapor contamination levels will not approach liquid contamination levels.

The estimates of skin LD_{50}s and skin LCt_{50}s associated with the peak contamination of casualties for each agent and each attack scenario are shown in Table 3.1. The maximum calculated toxicities were 67 LD_{50}s and 1.0 LCt_{50} for HD, 118 LD_{50}s and 0.8 LCt_{50} for GB, 57 LD_{50}s and ?LCt_{50}[3] for GD, 570 LD_{50}s and ?LCt_{50}[3] for TGD, 400 LD_{50}s and 0 LCt_{50} for VX, and 960 LD_{50}s and 0 LCt_{50} for TVX. It should be reiterated that inhalation toxicities are not considered in this contamination analysis because it is assumed that no respiratory activity is present in the dead casualty.

3.2.3 Whole-Body Immersion

The identification or definition of the worst-case exposure scenario has been debated, and some very extreme possibilities have been suggested. One of these is the complete immersion of a casualty or the remains in agent. Although complete immersion is not expected to be a commonplace occurrence, it could happen on the battlefield or as the result of an accident and thus deserves consideration. It is anticipated that with this scenario, the expected high residual agent levels in the body of the decontaminated remains would pose the greatest potential hazard to handlers.

A preliminary comparison of peak agent contamination levels generated by the explosive munitions (Section 3.2.1) with contamination levels due to complete immersion is shown in Table 3.2. It is assumed that complete immersion will leave a film of agent 0.1 mm thick on a skin surface of 2 m^2. The level of agent penetration is again expressed as the number of percutaneous LD_{50}s of liquid agent (Section 3.2.4). The analysis shows that for HD, GB, and TGD, the comparative agent levels per casualty are roughly equal for the two scenarios. The tenfold higher level calculated for GD contamination by immersion is probably due to the low levels of GD generated by the munition used. The major points to note are the large contamination and toxicity levels associated with VX exposure by immersion. The 36- to 54-fold increases in the number of VX and TVX LD_{50}s by immersion over those produced by munition-released agent is a clear reflection of the much greater percutaneous penetration of VX, as well as the low agent levels generated by the munitions used.

3.2.4 Wounds

The unclassified and readily accessible literature for the effects of wounds on nerve agent absorption and toxicity is comprehensively presented in an unclassified 1986 review (Augerson *et al.*, 1986) of the general toxicology and therapy for nerve agents. It has been reported that agent effective time is greatly reduced and may result in severe effects with "lightning speed" Augerson *et al.*, 1986). Even with HD, for which injury is characteristically delayed following topical exposure, contamination of wounds will speed the progression of systemic injuries. Just a few drops could result in death, even before skin vesication has developed at contaminated skin sites. These effects of wounds presumably reflect the degree to which removal of the skin barrier speeds agent absorption. Although the stratum corneum, lucidum, and outer epidermis are penetrated relatively rapidly by nerve agents, an intact lower epidermis provides a much more resistant barrier to nerve agent penetration. It was observed (Augerson *et al.*, 1986) that parenteral routes of administration (subcutaneous, intramuscular, intravenous and intra-arterial) may provide useful information as models for the effects of wounds on nerve agent toxicity and

[2]Dosage is the time-integrated concentration history, and its dimensions are mg·min/m^3.

[3]The skin LCt_{50} for GD is not known.

Table 3.2. Comparison of potential agent contamination levels by explosive battlefield munitions and by whole-body immersion in agent.

Agent	Explosive Munitions[a] g/casualty	LD_{50}/casualty[c]	Whole-Body Immersion[b] g/casualty[d]	LD_{50}/casualty[c]
HD	200	67	254	84
GB	200	118	220	130
GD	20	57	204	592
TGD	200	570	204	592
VX	4	400	216	21,600
TVX	6	960[e]	216	34,560[e]

[a]Data from Table 3.1. Peak liquid exposure levels.
[b]Assumes residual agent film, 0.1 mm thick, covering a skin area of 2 m^2. Total volume = 200 cm^3.
[c]Number of percutaneous LD_{50}s = g/casualty/g/human LD_{50}.
[d]g/casualty by whole-body immersion = 200 cm^3 x density of agent.
[e]Thickened VX is 1.6 times more toxic than neat VX (Tomlinson and Samuel, 1980).

toxicokinetics. In this regard, it has been reported that VX in humans is about 32 times more potent by the intravenous route than by percutaneous exposure in producing a 50% inhibition of erythrocyte acetylcholinesterase (1 µg/kg versus 32 µg/kg). Moreover, it has been estimated that the percutaneous toxicity of VX may be 100-150 times that of the G agents. This higher toxicity of VX is consistent with the observation that VX is not readily hydrolyzed in skin, and that it may persist in and distribute from skin for up to 24 hours.

The contamination of wounds in remains without a viable circulation can be expected to result in large local accumulation of agents. The potential hazard that such accumulation of agent would pose to handlers of the remains would depend on both the decontamination efficacy for wounds[4] and the detoxification rate of agents.

Table 3.3 presents estimates of the effects of wounds and damaged skin, relative to intact skin, on the amounts (LD_{50}/100 cm^2) of active agents that might enter the body of a casualty. In this analysis, absorption of agent

[4]Several studies have addressed the decontamination of chemically contaminated wounds. A recent report compared the decontamination efficacies of 0.5% and 5% NaOCl and $Ca(OCl)_2$ solutions against either VX or HD (Hobson and Snider, 1992). Three rabbit contamination models were used: (1) intact skin, (2) 8-mm-deep skin wounds made with a 5-mm diameter biopsy punch, and (3) contaminated swatches of winter-weight battle dress inserted into skin wounds. Decreases in 24-hour lethality (protective ratios) were determined from animals decontaminated with NaOCl after VX exposure (9 minimum lethal doses), and decreases in the size of dermal lesions caused by 0.5-µl exposure to HD were evaluated after decontamination with NaOCl or $Ca(OCl)_2$. The rates of decontamination of VX and HD on swatches were determined by analysis of agent remaining on the swatch at various times. The levels of VX and HD used in this study were much lower than those which might be expected in contaminated remains under worst-case scenario conditions. Although 0.5% NaOCl was almost as effective as 5% NaOCl as a decontaminant against topical VX exposure, it was only one-fifth as effective as 5% NaOCl against VX in the two dermal injury exposure scenarios. Only the 5% NaOCl concentration offered significant protection (protective ratios were 0.8 and 3.9 for 0.5% and 5% NaOCl, respectively). Although 5% $Ca(OCl)_2$ was more efficacious than the 0.5% solution if applied 3 minutes after HD exposure (an unlikely scenario with contaminated remains), the two concentrations of $Ca(OCl)_2$ were equally effective if applied after 5 minutes. On the other hand, 0.5% and 5% NaOCl concentrations had similar decontamination efficacies against HD skin contamination over exposure periods of 1-60 minutes. Both NaOCl solutions generally performed poorly in the decontamination of swatches, except that 5% NaOCl, if applied within 5 minutes of HD, reduced contamination by 50%. Thus 0.5% NaOCl was as effective as 5% NaOCl against topical exposures of HD, but neither concentration demonstrated sustained efficacy against HD on fabric in wounds. However, several classified reports indicated that simple modifications of the hypochlorite solution would result in significant improvement in the efficacy of decontamination of wounds containing VX, GD, HD, or THD.

Table 3.3. Estimates of the number of agent LD_{50}s available for absorption from intact skin, from damaged skin, and from 100-cm^2 wounds for various worst-case scenarios. Absorption from wounds is assumed to be by intramuscular and/or intravenous routes.

Agent	Maximum[a] Exposure Levels (g/man)	(mg/100 cm^2)	Dosage (LD_{50}/100 cm^2) Dermal[b] (Intact Skin)	Dermal[c] (Damaged Skin)	IM[d]	IV[e]
Explosive Munitions						
VX	4	20	2.0	1.9-9.5	24	36
GD	20	100	0.29	0.95-2.0	450	830
GB	200	1,000	0.59	4.8-9.5	—[f]	1,000
TVX	6	30	4.8	2.9-14.3	35	54
TGD	200	1,000	2.9	9.5-20.4	4,500	8,300
HD	200	1,000	0.33	—[f]	—[f]	—[f]
Whole-Body Immersion[g]						
VX	216	1,080	108	103-510	1,300	1,900
GD	204	1,020	3.0	9.7-20.8	4,600	8,500
GB	220	1,100	0.65	5.2-10.5	—[f]	1,100
TVX	216	1,080	108	103-510	1,300	1,900
TGD	204	1,020	3.0	9.7-20.8	4,600	8,500
HD	254	1,270	0.42	—[f]	—[f]	—[f]

[a]Source: Table 3.1.
[b]Intact skin LD_{50}s for VX, GD, GB, and HD are 10, 350, 1,700, and 3,000 mg/man, respectively (see Table 3.1).
[c]Damaged skin LD_{50}s for VX, GD, and GB are 2.1-10.5, 49-105, and 105-210 mg/man, respectively (Augerson *et al.*, 1986).
[d]IM LD_{50}s for VX and GD are 0.85 and 0.22 mg/man, respectively (Anderson, 1974; Facklam, 1982).
[e]IV LD_{50}s for VX, GD, and GB are 0.56, 0.12, and 1.0 mg/man, respectively (Anderson, 1974; Facklam, 1982).
[f]LD_{50} values not available.
[g]Assumes residual agent film, 0.1 mm thick, covering a skin area of 2 m^2; total volume = 200 cm^3; g/man by whole-body immersion = 200 cm^3 x density of agent.

from wounds is assumed to be by the intramuscular and/or intravenous routes. Estimated LD_{50}s for humans were used in the calculations.

The data in Table 3.3 represent worst-case exposures in several respects. The estimates were derived for severe exposure scenarios-exposure to explosive munitions and exposure by whole-body immersion. Moreover, it was assumed that no decontamination was performed, and that all of the agent on the 100-cm^2 body surfaces was absorbed by the respective routes (dermal for intact and abraded skin; intramuscular and intravenous for wounds).

Our analysis assumes that the amounts of agent absorbed by wounds of increasing severity are represented by the progression of data for intact skin < damaged skin < i.m. route < i.v. route. This assumption appears to be validated: in every case for which data are available, absorption (LD_{50}s) increases progressively in the order intact skin, damaged skin, intramuscular route, intravenous route. The most notable finding is that the amount of agent that could be absorbed for the nerve agents (data not available for HD) progressively increases much more markedly with increasing wound severity for G agents than for VX or TVX. For example, with explosive munitions, the ratios obtained by dividing the available dosage (LD_{50}s) with the intravenous route by that for intact skin are 2,900 and 1,700 for GD and GB, respectively, compared to 18 for VX. Similarly, for whole-body immersion, these ratios are 2,800 and 1,700 for GD and GB, respectively, and only 18 for VX.

The analysis indicated in Table 3.3 suggests that wounds markedly increase the absorption of GD and GB, while having a much lesser effect on absorption of VX, which has an inherently higher and sustained percutaneous penetration. Nevertheless, these data do not necessarily indicate a major increase in the hazard of G agents to handlers, primarily because G agent contamination is reduced as a result of rapid spontaneous and enzymatic hydrolysis (see Section 3.5).

It was also noted that sequestration of agent might result from clothing, projectiles, shrapnel, or other objects that might be found in a wound. These objects should be removed from the wound and decontaminated separately.

3.3 FATE OF THREAT AGENTS IN THE BODY

This section provides a brief overview of some chemical and physical properties of threat agents pertinent to their fate before, during, and after absorption into the body. *In vivo* detoxification of agents by hydrolysis, especially that due to enzymatic hydrolysis, stereoselective reaction and hydrolysis, excretory processes, and related issues are considered. This analysis generally leads to the conclusion that at the levels of battlefield contamination indicated for the worst-case scenarios discussed in Section 3.2, all enzyme phosphonylation and nonspecific binding sites for nerve agents will be completely saturated. This is not the case for sulfur mustard (HD), as there are almost an infinite number of nonspecific binding sites for this vesicant. Thus, for the nerve agents, very large exposures will leave excess "free" agent that could potentially pose a hazard to handlers. The excess free agent will be subject to spontaneous and/or enzymatic hydrolysis. It is not clear how these metabolism activities will impact on detoxification of agent in the remains.

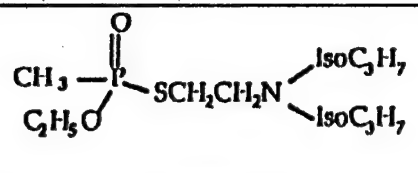
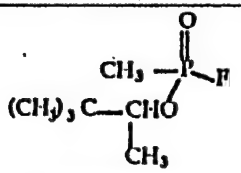
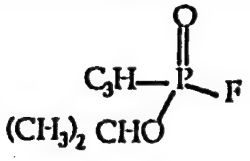
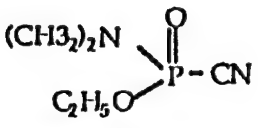
Based on the following consideration of factors related to the fate of threat agents in the body, it is concluded that G agents and HD will be detoxified in the decontaminated body and will not pose a hazard. Only VX could pose a hazard to handlers.

3.3.1 General Properties of Threat Agent

Since this section is provided only as a brief overview of relevant chemical, physical, and biological properties of agents, it will focus on key points depicted in Table 3.4.

Two pertinent issues arise from the structures of nerve agents: (1) the existence of stereoisomers and (2) susceptibility of the agents or their isomers to enzymatic hydrolysis by phosphonylphosphatases. This class of enzymes was referred to in early literature as individual enzymes such as somanase, sarinase, tabunase or DFPase (this nomenclature may still be used). However, the existence of individual specificities of the latter enzymes for a single agent or organophosphorus compound is highly suspect (Reiner *et al.*, 1989). The properties of the stereoisomers of these nerve agents, particularly GD, have recently been reviewed and analyzed (Benschop *et al.*, 1984a; Benschop and De Jong, 1988; Benschop *et al.*, 1984b). Each of the four nerve agents in Table 3.4 consists of stereoisomers that are present in equal amounts. Racemic GD consists of four stereoisomers, while the remaining nerve agents contain only two stereoisomers. The source of two isomers for each nerve agent is the center of asymmetry at the stereogenic phosphorus atom. For GD, two additional stereoisomers are formed due to the chiral center in the pinacolyl moiety. The primary importance of this isomerism lies in stereoselectivity of action of these isomers, resulting in varying toxicities, different rates of hydrolysis and other reactions, and differences in agent toxicokinetics and toxicodynamics.

Table 3.4. General properties of threat agents.

	VX	GD (Soman)	GB (Sarin)	GA (Tabun)	HD
Structure					
Rate of Spontaneous H_2O Hydrolysis[a] (General)	Varies with pH[b]	Varies with pH[b]	Very rapid in alkaline solutions. In acid conditions destroyed in lab within 15 hours.[b]	Slow with distilled water at pH 7.0 but increasingly rapid with any acid or alkali present.[b]	Not pH-dependent[d] Rapid[b]
Water Solubility	3.0 g/100 g (25°C)[d,e]	2.1 g/100 g (20°C)[f,g]	Miscible[b] (completely)[h]	9.8 g/100 g (25°C)[i]	0.092 g/100 g (22°C)[i,g]
Volatility[j]	10.5 mg/m^3 (25°C)[b,k]	3,900 mg/m^3 (25°C)[b]	22,000 mg/m^3 (25°C)[b]	610 mg/m^3 (25°C)[b]	920 mg/m^3 (25°C)[i,g]
Vapor Pressure	0.0007 mmHg (25°C)[b,k]	0.4 mmHg (25°C)[b]	2.9 mmHg (25°C)[b]	0.07 mmHg (25°C) (low)[b]	0.11 mmHg (25°C)[i]
Vapor Density	9.2 x air[b,g]	6.33 x air[b]	4.86 x air[b]	5.63 x air[c]	5.4 x air[c]
Liquid Density	1.008 g/cc (25°C)[b,k]	1.022 g/cc (25°C)[b]	1.09 g/cc (25°C)[b]	1.073 g/cc (25°C)[b]	1.27 g/cc (25°C)[i]
Persistency[l] (General)	Highly persistent[b] Persistent[m]	Mildly persistent[b] Nonpersistent[m]	Essentially nonpersistent[b] Nonpersistent[m,b]	Nonpersistent[m]	Persistent[m]
Rate of Detoxification (General)	Very slow[b]	Slow[b]	Slow[b]	Slow[b]	Very slow[b]

[a]For more specific data, see Table 3.5.
[b]Compton, 1987.
[c]Compton, 1987.
[d]Tomlinson and Samuel, 1980.
[e]Yurow and Davis, 1982.
[f]Gum *et al.*, 1992.
[g]Fargo *et al.*, 1988.
[h]Yurow, 1981.
[i]Anderson, 1974.
[j]Volatility is generally directly related to its rate of evaporation from human skin. For example, VX by far has the slowest evaporation rate from human skin (Compton, 1987). Similarly, evaporation of tabun droplets from skin of humans occurs in 1/2 to 3 hours under meteorological conditions favoring the evaporation of sarin from 2 to 13 minutes (Freeman *et al.*, 1954).
[k]Kalkwarf *et al.*, 1987.
[l]Persistency here specifically refers to that in the environment (e.g., soil) and not to that in biological organisms.
[m]Author unknown, 1981.

Phosphonylphosphatases are hydrolytic enzymes widely found in diverse tissues of various mammalian species, including humans. The enzymes hydrolyze the nerve agents GD, GB, and GA by attacking phosphorus ester groups (O'Brien, 1960). However, the more complex structure of VX apparently renders it not susceptible to attack by this class of enzymes (Scaife and Campbell, 1959; O'Brien, 1960). It has been proposed that enzymatic metabolism and detoxification of VX and similar structures require oxidation (Scaife and Campbell, 1959; Harris *et al.*, 1984), and that after oxidation, the compounds may be rendered very susceptible to enzymatic hydrolysis (O'Brien, 1960). However, this oxidative enzyme system proposed for VX and some structurally related compounds has not been identified in humans (Scaife and Campbell, 1959).

Spontaneous water hydrolysis and water solubility are considered in some detail in Sections 3.3.2 and 3.3.4.1, and are only mentioned here. Of those agents considered, the spontaneous hydrolysis of the nerve agents depends on both pH and temperature, while that for HD is a function only of temperature. Moreover, HD dissolved in aqueous media generally is the most rapidly hydrolyzed agent. HD is very sparingly soluble in water, GB is completely miscible, and the remaining three nerve agents have similar solubilities ranging from 2.1-9.8 g/100 g at 25°C.

Table 3.4 indicates that VX has the lowest vapor pressure and volatility of the agents studied, along with the highest vapor density. Therefore, it is not surprising that VX has by far the slowest rate of evaporation from human skin (Compton, 1987).

The detoxification rate of VX has been reported to be very slow (Compton, 1987). The data in Table 3.4 indicate that the G agents are more rapidly detoxified in the body than is VX. HD is generally considered to have a very slow detoxification rate (Compton, 1987). However, when HD is dissolved in water under physiological conditions in the body, it may undergo rapid detoxification as a result of hydrolysis.

3.3.2 Spontaneous Hydrolysis of Threat Agents in Water

Water hydrolyzes nerve agents by nucleophilic reaction at the phosphorus bond (i.e., P-X) as indicated in Table 3.4. The X atoms or chemical groups are F for GB and GD, CN for GA, and $SCH_2CH_2N[CH(CH_3)_2]_2$ for VX. The rate of spontaneous water hydrolysis is a function of both temperature and pH. Unfortunately, although rates of water hydrolysis of the different nerve agents are frequently reported in the literature, they are often provided with little or no experimental details and commonly lack the original citation. This may explain what appear to be frequent contradictory values for hydrolysis rates, particularly for VX. For example, the half-life of VX in water at 25°C and pH 7.0 is reported to be 966 hours in many sources as compared to 40 hours in others. The shorter half-lives in the literature are generally better substantiated in that they were determined under experimental conditions that better approximate physiological conditions. For example, buffer systems similar to those found in the body may increase hydrolysis rates for GB and GD by 6- to 7-fold compared to the rates determined in unbuffered aqueous solutions (Ellin *et al.*, 1981). Similar criteria for credibility were used in selecting the values presented in Table 3.5.

For ease and practicality of comparison of the hydrolysis rates in water, a temperature of 20°-25°C and pH of 6.65-7.0 were selected. At these conditions, the half-lives in water are rather slow and are similar for VX (40 hours), GD (45 hours), and GB (46 hours) (Table 3.5). On the other hand, the half-life of GA under comparable conditions is substantially shorter at 8.5 hours. It has been noted consistently that in human body fluids with pH values from 7.34 to 7.42, the half-life for VX is about 40 hours and that for GD is about 45 hours (Metz *et al.*, 1988). The few values provided here for seawater with GD, GA, and HD suggest that this medium decreases half-life for the G agents and increases half-life for HD.

Sulfur mustard dissolved in water rapidly hydrolyzes independently of pH with a half-life of 2.6 minutes at 37.4°C. Since HD is only sparingly soluble in water, the overall hydrolysis rate will be dependent on its rate of solution. In dilute aqueous solution, HD hydrolyzes almost completely in two stages to hydrochloric acid and thiodiglycol (TDG), reactions which are mediated by the rate-limiting formation of highly reactive cyclic ethylene sulfonium intermediates. Due to the reversibility of the activation steps, chloride ions present in physiological environments will increase the half-life of hydrolysis about fourfold (Papirmeister *et al.*, 1991).

3.3.3 Detoxification of Sulfur Mustard (HD) in the Body

3.3.3.1 General

The World War II literature on the detoxification of HD has been recently reviewed (Renshaw, 1992). In brief, the major exposure routes for HD vapor or liquid result in absorption via the skin and eyes, while vapor

Table 3.5. Spontaneous hydrolysis of threat agents in water.

Medium	VX °C	VX pH	VX Half-life	GD (Soman) °C	GD (Soman) pH	GD (Soman) Half-life	GB (Sarin) °C	GB (Sarin) pH	GB (Sarin) Half-life	GA (Tabun) °C	GA (Tabun) pH	GA (Tabun) Half-life	HD (Sulfur Mustard) °C	HD (Sulfur Mustard) pH	HD (Sulfur Mustard) Half-life[a]
Water				25	2	3 hr[b]	0	7.5	837 hr[b]	20	4-5	7 hr[c]	0.6	-	158.0 min[d]
				25	5	41 hr[b]	4	7.5	453 hr[b]	20	7	8.5 hr[c]	10.0	-	57.8 min[d]
				25	6.65	45 hr[b]	20	7.0	46 hr[b]				20.0	-	14.7 min[d]
				25	8	50 hr[b]	25	7.5	24 hr[b]				30.0	-	3.3 min[d]
				25	10	60 hr[b]							37.4	-	2.6 min[d]
	25	2-3	100 day[c]				25	1.8	7.5 hr[e]						
	25	7	40 hr[c]				25	6.0	47 hr[e]				0.0	-	105.0 min[f]
	25	11	17 hr[c]				25	9.0	5 hr[e]				25.0	-	4.0 min[f]
	25	12	2 hr[c]				25	10.9	4.2 hr[e]				40.0	-	0.72 min[f]
	25	13	0.2 hr[c]												
	25	14	1.3 min[c]												
Seawater				25	-	8 hr[g]				15	-	7.9 hr[g]	25.0	-	60.0 min[d]
										20	-	4.45 hr[g]	30.0	-	25.0 min[d]
										25	-	2.9 hr[g]			
Skin[h]				37	7.5	7.5 hr[i]	37	7.5	3.8 hr[i]	37	7.5	6.75 hr[i]			

[a]The chloride present at physiological conditions would increase the half-life approximately fourfold.
[b]Fargo *et al.*, 1988.
[c]Weyandt, 1991.
[d]Papirmeister *et al.*, 1991.
[e]Anderson, 1974.
[f]Penski, 1983.
[g]Yurow and Davis, 1982.
[h]Guinea pig skin suspension heated at 90°C for 25 min to inactivate enzymes possibly present, and dissolved in 0.1 M KCl.
[i]Fredriksson, 1969a.

enters through the respiratory tract epithelium. For percutaneous exposure of the total quantity of HD that contacts the skin, only a relatively small amount is actually absorbed. For instance, it has been found that when a small drop of liquid HD is applied to the skin at a temperature (22°C), humidity, and wind speed within a typical environmental range, about 80% evaporates and 20% penetrates the skin. Even after exposure to high concentrations of HD for short periods, only a small percentage of the absorbed dose alkylates tissue around the absorption site. Most of the HD is absorbed into the blood as unreacted or hydrolyzed HD (or as metabolites), which is rapidly distributed to all major organs, possibly excepting the eye. The kidney most highly concentrates HD or its metabolites, and high levels are also found in liver and intestines.

Studies on the metabolism of HD, primarily with rats, are very limited (Papirmeister *et al.*, 1991). Alhough the evidence is not conclusive, it appears that the two most important routes of detoxification are the hydrolysis of HD to form TDG and the reaction of HD with glutathione. Although possible enzymatic detoxification has not been definitively ruled out, most of the evidence indicates that the major metabolites of HD are formed through direct alkylation reactions. The major route of excretion for HD and its metabolites appears by far to be renal, although biliary excretion may be a significant but lesser route. After intravenous administration of radiolabeled HD in rodents, essentially all excreted radioactivity was present in the urine. Approximately 80% of the injected radioactivity was excreted within the first 24 hours, most within the first 6 hours.

3.3.3.2 Possible Sulfur Mustard Depots

It has been suggested that unchanged HD, HD as a toxic metabolite, or HD in potentially toxic form persists in the human body for extended periods (hours and days) after HD exposure (Papirmeister *et al.*, 1991). However, the extended presence of any of these HD forms remains to be proved. Suggestions of persistence are derived primarily from a few studies of casualties of the Iran-Iraq War and from *in vitro* experiments demonstrating the formation of more complex sulfonium salts from HD and TDG. It has been suggested that under certain conditions, sulfonium salts may be formed from the reaction of HD and TDG *in vivo*. Moreover, it was proposed that the formation and decomposition of sulfonium salts *in vivo* may have resulted in the relatively long-lasting presence of HD or HD biotransformation products in the urine of several war casualties and in other body tissues of a single casualty. However, the formation of sulfonium salts has not been demonstrated *in vivo*, and the concentrations of HD and TDG required for such formation are so high that their occurrence in the body is unlikely. Furthermore, the presence *in vivo* of compounds with competition factors higher than TDG does not favor the formation of sulfonium salts. Finally, only TDG, and not HD or other HD metabolites, was isolated from the urine of casualties. Small concentrations of TDG may be found in the urine of humans that have not been exposed to HD. Since TDG is a major, nontoxic metabolite of HD, the approximately week-long presence of small amounts of TDG reported for a small number of casualties of the Iran-Iraq War seems weak evidence for any significant HD depot.

3.3.4 Detoxification of Nerve Agents in the Body

The focus of this section on nerve agent detoxification will be to present human *in vivo* data, as they are certainly the most important and relevant data for the objectives of this report. If support for this approach is needed, a comprehensive review of the toxicology of nerve agents in various species noted that preclinical toxicology data derived from animal experiments are seldom directly applicable to humans (Facklam, 1982). However, pertinent human *in vivo* data are often lacking due to specific properties of the nerve agents, such as high toxicity, relatively irreversible effects (e.g., aging of acetylcholinesterase, particularly on reaction with soman), and the difficulty of quantifying enzyme kinetic data *in vivo* (Maxwell *et al.*, 1988; Maxwell *et al.*, 1987; Facklam, 1982). Furthermore, the *in vivo* kinetics for the inhibition of cholinesterase by nerve agents, as well as the biochemical and physiological factors that influence these reactions, are poorly understood (Van Dongen *et al.*, 1986). Thus, in several instances, *in vitro* enzyme kinetics are presented for ease of quantification and comparison of nerve agents, and some animal studies are considered. Important cases for which human data are lacking are indicated in Section 3.3.4.7.

3.3.4.1 Skin Detoxification: Spontaneous Water and Enzymatic Degradation

A recent review (Metz *et al.*, 1988) referred to studies conducted in the early 1960s with VX and some G agents (GD, GB, and GF) to determine their evaporation rates from excised pig skin at 32°C and from glass. In additional experiments on evaporation rate, a drop of these G agents (1-2 mg) was placed on pig

skin in a wind tunnel, and air was drawn through at a constant velocity of 1m/sec. The initial evaporation rates from skin and glass were similar for all agents. However, VX only partially evaporated while almost all of the two G agents evaporated. VX evaporated at a steadily decreasing rate that was proportional to the saturation vapor concentration of the skin surface, while G agents evaporated at an approximately constant rate. The order and estimated half-lives for evaporation from pig skin were GB (2 minutes) <GD (4 minutes) <VX (17 hours).

The rate of detoxification of nerve agents within the skin is a function of spontaneous water hydrolysis and enzymatic hydrolysis by phosphonylphosphatases (possible agent depots from percutaneous exposure are discussed in Section 3.3.4.4). As noted above, while G agents are hydrolyzed by phosphonylphosphatases, VX is not, and spontaneous hydrolysis in water for all these cholinesterase inhibitors is slow. Relevant here is a report dealing with studies of VX penetration through the skin of rabbits *in vivo* (Marzulli and Wiles, 1957). It was concluded that VX apparently penetrated the skin into the bloodstream without significant hydrolysis or retention by tissues. On the other hand, experiments on the penetration of VX applied to the forearm of human volunteers (Cummings and Craig, 1965) indicated that VX accumulated in the epidermis at lower temperatures and was not susceptible to surface decontamination by bleach. Although it subsequently entered the body when blood circulation was restored by return of the exposed volunteer to room temperature, the agent may remain sequestered in the epidermis of a dead casualty.

Two additional reports are particularly pertinent to the skin detoxification of G agents, and their results, together with other information, may allow inferences about VX. In these studies with GD, GA, and GB, rates of penetration through cat skin *in vivo* (Fredriksson, 1969b) and hydrolysis rates in guinea pig skin suspensions *in vitro* (Fredriksson, 1969a) were determined. Data from the latter study, for spontaneous hydrolysis (heated skin suspension), are contained in Table 3.5, and those for enzymatic hydrolysis, in Table 3.6. The results of the two studies are generally consistent. For GD the rate of enzymatic hydrolysis in guinea pig skin was particularly fast, with a half-life of 47.5 minutes, while GB and GA had similar values of 125 and 130 minutes, respectively. On the other hand, for spontaneous hydrolysis, GB had the fastest half-life, 230 minutes, while those of GA (405 minutes) and GD (461 minutes) were each about twice as slow as that of GB. The study with cats *in vivo*, in which cardiac arrest was used as an indirect measure of agent absorption rates ($\mu M \cdot min^{-1} \cdot cm^{-2}$), found that GD (which had the highest rate of enzymatic hydrolysis in guinea pig skin suspension) had the slowest rate of skin absorption, 1.5, while higher penetration rates occurred for GA (4.8) and GB (10.5).

Based on the absence of phosphonylphosphatase hydrolysis of VX and the previous estimate of similar rates of spontaneous water hydrolysis for VX and these three G agents, it appears that VX would be the most rapidly absorbed of these nerve agents. Moreover, data for evaporation rates from skin indicate that VX would be absorbed over the longest period of time under similar exposure conditions.

3.3.4.2 Toxicity, Enzymatic Hydrolysis, and Stereoselectivity of Nerve Agents

It has long been accepted that the major toxicity of nerve agents, including VX and the G agents considered in this report, can be attributed to the irreversible inhibition of acetylcholinesterase (AChE; Koelle, 1981; Richardson *et al.*, 1976; Tomlinson and Samuel, 1980; Harris, 1945; Facklam, 1982). The inhibited acetylcholinesterase may also undergo a second process called "aging," which is particularly fast for soman. Aging makes reactivation of the enzyme very difficult; for soman, aging is essentially complete within 10 minutes after initial inhibition (Somani *et al.*, 1992). There is also much long-standing evidence that GA, GB, and GD are detoxified in the body through hydrolysis by phosphonylphosphatases found in diverse tissues and organs of humans and other species (O'Brien, 1960; Hoskin, 1971; Augustinsson and Heimbürger, 1954a; Augustinsson and Heimbürger, 1954b; Augustinsson, 1957; Augustinsson and Heimbürger, 1954c; Augustinsson and Heimbürger, 1954d; Boter and Van Dijk, 1969; Keijer and Wolring, 1969). Because of its importance in this report, it is emphasized again (also see Section 3.3.4.1) that VX is not enzymatically hydrolyzed by phosphonylphosphatases or other known enzymes. Specifically, there is no evidence to support suggestions that VX and structurally related compounds are degraded by oxidation in the human body (Scaife and Campbell, 1959; Harris *et al.*, 1984; O'Brien, 1960).

It can be seen from the bimolecular rate constants for AChE in Table 3.6 that the racemic forms of VX and the G agents generally react at a similar, rapid rate with AChE. The single apparent exception is GA, which inhibits AChE at a substantially slower rate than do racemic mixtures of the other agents assessed. However, *in vivo* evidence indicates that VX has greater specificity for AChE than do GD, GB, and GA; this study demonstrated that VX has relatively poor affinity for nonspecific binding sites (Boskovic, 1979). Similarly, in an *in vitro* enzyme kinetic study, VX when compared to

Table 3.6. Rate of reaction/hydrolysis of threat agents *in vitro*.

	Acetylcholinesterase (AChE)			Phosphonylphosphatase[a]			Butyrylcholinesterase (BuChE)[b]			Carboxylesterase (CaE)[c]		
	Human RBC	Bovine RBC	Eel	Human Serum	Bovine Serum	G. Pig Skin	Human Serum	Horse Serum	Dog Serum	Human Plasma	Rat Plasma	Rat Liver
	Bimolecular Rate Constant (M^{-1} min^{-1})			Rate Constant (min^{-1})		Half-Life (min)	Bimolecular Rate Constant (M^{-1} min^{-1})			Bimolecular Rate Constant (M^{-1} min^{-1})		
VX												
(±)	$1.6X10^7$ [d]	$5.0X10^7$ [e]	$6X10^7$ [f]							ND[t, g]	$1.5X10^3$ [h]	$1.5X10^3$ [i]
(-)		$4X10^8$ [j]								ND[t, g]		
(+)		$2X10^6$ [j]								ND[t, g]		
GD												
C(±)P(±)		$1.7X10^7$ [f]	$6.4X10^7$ [f]			47.5[k]				ND[t, g]	$5.1X10^6$	$5.2X10^6$
C(-)P(-)		$2.70X10^7$ [m]	$1.8X10^8$ [m]	0.01288	0.00798		$0.61X10^7$	$0.64X10^7$ [l]	$6.6X10^5$ [o]	ND[t, g]		
C(+)P(-)		$1.75X10^8$ [m]	$2.8X10^8$ [m]	0.00508	0.00558		$3.6X10^7$ [o]	$8.5X10^7$ [l]	$7.2X10^6$ [o]	ND[t, g]		
C(-)P(+)		$<4X10^4$ [m]	$<5X10^3$ [m]	0.448	0.308		$3.3X10^5$ [p]	$6.4X10^6$ [l]	$4.0X10^5$ [o]	ND[t, g]		
C(+)P(+)		$<4X10^4$ [m]	$<5X10^3$ [m]	4.28	3.38		$1.2X10^5$ [p]	$2.9X10^5$ [l]	$2.7X10^7$ [o]	ND[t, g]		
GB												
(±)	$1.5X10^7$ [d]	$2.7X10^8$ [f]	$1.2X10^7$ [f]			125[k]		~$6X10^6$ [p]		ND[t, g]	$3.0X10^6$	$3.1X10^6$
(-)		$1.4X10^7$ [p]						~$6X10^6$ [p]		ND[t, g]		
(+)		$3.3X10^3$ [p]						~$6X10^6$ [p]		ND[t, g]		
GA												
6(±)			$1.3X10^5$ [r]			130[k]				ND[t, g]	$2.4X10^6$	$2.2X10^6$
(-)			$2.6X10^6$ [q]							ND[t, g]		
(+)			$3.7X10^5$ [q]							ND[t, g]		

[t]Not determined.
[a]Phosphonylphosphatases hydrolyze all G agents, but not VX, in a wide variety of mammalian species, including humans (e.g., Harris *et al.*, 1984; Fecklam, 1982).
[b]Butyrylcholinesterases react both with all G agents and with VX in a wide variety of mammalian species, including humans (e.g., Maxwell *et al.*, 1991; Keijer and Wolring, 1969).
[c]Carboxylesterases, when present in blood and other tissues, react with both G agents but have low affinity for VX. However, CaE is present in mammalian species in a phylogenetic manner, such that the relative amount of CaE in blood is zero in humans and rhesus monkeys, minor in marmosets, medium in guinea pigs, and large in rats (Van Den Berg *et al.*, 1984; Satoh, 1987; Reiner *et al.*, 1989).
[d]McNamara *et al.*, 1973.
[e]Mounter and Ormston, 1967.
[f]Mitz *et al.*, 1966.
[g]Reiner *et al.*, 1989.
[h]Maxwell, 1992.
[i]Maxwell, 1989.
[j]Benschop and De Jong, 1988.
[k]Fredriksson, 1969a.
[l]Keijer and Wolring, 1969.
[m]Benschop *et al.*, 1984a.
[n]De Bisschop *et al.*, 1987.
[o]Maxwell *et al.*, 1991.
[p]Boter and Van Dijk, 1969.
[q]Degenhardt *et al.*, 1986.
[r]Hoskin, 1971.

soman and sarin, was found to have the fastest rate of AChE phosphonylation (Forsberg and Puu, 1984).

For obvious reasons, systematic experiments in which AChE activity is measured after exposure to high levels of nerve agents have not been done in humans. A review presents results of experiments in which human volunteers were exposed to relatively low levels of nerve agents. Case reports in which personnel were accidentally exposed to high but nonlethal doses were also presented (Facklam, 1982). In both types of exposures, the nerve agents were VX, GA, GB, and GD. Even low-level exposure (as accessed by overt and physiological measures) often produced marked reductions in AChE levels in blood, while high-level exposure nearly or completely abolished AChE activity. Thus it can be expected that for the worst-case scenarios considered here, all AChE binding sites will be saturated by nerve agent. On the other hand, since phosphonylphosphatases catalyze the hydrolysis of G agents but do not react irreversibly with them (O'Brien, 1960; Reiner *et al.*, 1989), these enzymes should not be similarly saturated.

The isomers of nerve agents show various degrees of stereoselective reactions with these three enzymatic processes: (1) AChE inhibition, (2) aging of agent-inhibited AChE, and (3) hydrolysis by phosphonylphosphatases (except for VX, which does not react with these enzymes). Table 3.6 indicates that for the four agents (VX, GD, GB, GA) for which data on the isomers are available, the (-) form or forms (GD) at the phosphorus atom are always the most active inhibitors of AChE. From the differences in inhibitory activity (bimolecular rate constants) between the stereoisomers, the four agents may be distinguished into two groups (Benschop and De Jong, 1988): (1) GD and GB (3-4-order-of-magnitude difference), and (2) VX and GA (1-2-order-of-magnitude difference). For the highly potent AChE inhibitor VX (about 1-order-of-magnitude difference), the relatively small difference in the AChE affinities of its two stereoisomers appears to be a minimal confounding factor. Similarly, there is accumulating evidence that the (-) forms of the stereoisomers preferentially age AChE at the faster rate (Benschop *et al.*, 1984a; Keijer and Wolring, 1969).

Phosphonylphosphatases are generally considered to hydrolyze the isomers of GD, GB, and GA in a stereoselective manner (Benschop and De Jong, 1988; (Somani *et al.*, 1992). This stereoselectivity has been thoroughly demonstrated for GD in various mammalian species, including humans, for *in vitro* plasma and liver homogenates (Reiner *et al.*, 1989). All enzyme preparations hydrolyzed the soman stereoisomers in the order C(+)P(+)>C(-)P(+)>>C(-)P (-)>C(+)P(-). The reaction rates in human plasma are very similar to serum values given in Table 3.6. In a similar manner, the (+) isomer of GB is preferentially hydrolyzed *in vitro* in plasma of humans and various other species (Cohen *et al.*, 1971; Christen and Van den Muysenberg, 1965). The stereoselectivity of GA isomers, however, appears to be species-dependent. Phosphonylphosphatases from human plasma and from several other sources (e.g., porcine and murine plasma) preferentially hydrolyze the (+) form of GA, but enzymes from equine and bovine plasma have the reverse stereoselectivity (Benschop and De Jong, 1988). Recently, a phosphonylphosphatase has been partially characterized from rat liver and is reported to hydrolyze all four isomers of soman at approximately the same rate (Little *et al.*, 1989). The full characterization and significance of this enzyme remain to be determined.

3.3.4.3 Detoxification by Enzymatic Phosphonylation and Nonspecific Binding Sites

The two nonspecific enzymes with which nerve agents may react in the human body and be detoxified are butyrylcholinesterase (BuChE) and carboxylesterase (CaE). Various biological and toxicological properties of BuChE (Brown *et al.*, 1981; Reiner *et al.*, 1989) and CaE (Maxwell, 1992; Heymann 1980; Junge *et al.*, 1974; Satoh, 1987; Junge and Krisch, 1975) have recently been reviewed. In brief, both BuChE and CaE react stoichiometrically and irreversibly with all nerve agents studied (VX, GA, GB, and GD) in a manner analogous to that between nerve agents and AChE. However, the inhibition of BuChE and CaE by nerve agents produces no toxic effect. Although BuChE reacts with nerve agents at relatively high rates (see Table 3.6 for results with GD and GB in human and/or horse serum), BuChE, because of its low levels, should not be a significant means of nerve agent detoxification in the human body. For example, in mammals the BuChE levels are about one-thousandth of those for CaE (Maxwell *et al.*, 1987). Similarly, CaE should play only a minor role in the detoxification of nerve agents in humans, since only low levels are found in man, with none detected in human plasma (Reiner *et al.*, 1989). Moreover, VX has a relatively low rate of reaction with CaE. A study comparing the kinetics of VX, GD, GB, and GA found that VX was at least 500-fold less reactive *in vitro* with CaE than were the other agents (Maxwell, 1992). A primary factor proposed for the low affinity of VX for CaE is the quaternary nitrogen group in this nerve agent (Fonnum and Sterri, 1981).

The nerve agents considered in this report probably bind to nonspecific binding sites such as chymotrypsin

and trypsin (Somani *et al.*, 1992). However, the presence of these nonspecific binding sites is likely of little importance, particularly in the worst-case scenario, due to the relatively low affinity of nerve agents for these sites. This may especially be the case for VX, which has been reported to have a relatively poor affinity for nonspecific binding sites when compared with GD, GB, and GA *in vivo* (Boskovic, 1979). In summary, BuChE, CaE, and nonspecific binding sites will be completely saturated by nerve agents in the worst-case scenario.

3.3.4.4 Possible Nerve Agent Depots and Relationship to Temperature and Blood Flow

Two types of experiments have led to the proposal that nerve agent depots exist: (1) studies of anesthetized, atropinized, and artificially ventilated animals (including rats, guinea pigs, and marmosets) parenterally administered high doses (1-10 LD_{50}) of soman; and (2) percutaneous exposure, without therapy, of human skin to relatively low doses of nerve agents, particularly VX, at varying environmental temperature and skin site (e.g., cheek or forearm). The first type, in which animals and high doses of soman were used, involves a large and ongoing literature that has recently been reviewed (Van Helden *et al.*, 1984; Clement, 1984a; Somani *et al.*, 1992). The second type, usually with human volunteers, consists of a smaller number of studies, largely conducted in the mid-1960s (Cummings and Craig, 1965; Craig *et al.*, 1967) and published in the mid-1970s (Craig *et al.*, 1977a; Craig *et al.*, 1977b).

At the present time, the evidence for GD depots derived from the first type of experiment probably can be dismissed, especially when only the worst-case scenarios are considered. This is due to several factors: (1) GD is readily hydrolyzed by phosphonylphosphatases in humans (see, however, discussion on low rates of enzymatic detoxification of toxic isomers of G agents in Section 3.3.4.2). (2) The animals were exposed under artificial conditions inasmuch as they were anesthetized, atropinized, and artificially ventilated. (3) The GD depot is phylogenetically correlated, decreasing in magnitude from rats, to guinea pigs, to marmosets (Van Helden *et al.*, 1988). (4) The toxic signs attributed to release of the agent from the depot appear to last less than 24 hours after initial agent exposure. (5) It has been speculated, but not proved, that the site of the depot is plasma carboxylesterase (Clement, 1984b; Clement, 1982), muscle (Van Helden *et al.*, 1988; Van Helden and Wolthuis, 1983), adipose tissue (Sterri *et al.*, 1980), plasma cholinesterase (Sterri, 1981), and lung or skin tissue (Kadar *et al.*, 1985). Depot sites such as these may represent such a dispersion and/or dilution of the agent that much of it may be hydrolyzed on its release.

On the other hand, percutaneous exposure of humans to VX may result in agent depots within the skin that will require special precautions by handlers. This risk may apply particularly when human skin is initially exposed to VX at a high temperature, followed after a variable interval by skin decontamination, and the skin is then maintained at a relatively low temperature. When this sequence of conditions occurs and the final temperature is high, a subsequent decrease in erythrocyte AChE is observed, suggesting that VX was sequestered in the skin and released when the skin temperature was raised. However, depot formation may also occur when the casualty is initially exposed at a relatively low temperature and the body is maintained at a low temperature. A skin depot for VX, unlike that for G agents, would not be affected by phosphonylphosphatase. Skin temperature is highly correlated with skin blood flow. The skin is divided functionally, as opposed to anatomically, into an outer layer, accessible to blood flow and decontamination, and an inner layer, not susceptible to decontamination or possessed of blood vasculature. Thus, at the change to low temperatures, this inner layer may sequester agent, since it is not readily dispersed by blood flow (Cummings and Craig, 1965; Craig *et al.*, 1967; Craig *et al.*, 1977a; Craig *et al.*, 1977b; Sim, 1962; Lubash and Clark, 1960).

3.3.4.5 Excretion

Although elimination of nerve agents from the living body appears to have received little study (Hobson and Snider, 1992; Somani *et al.*, 1992), renal and, to a small degree, biliary excretions are considered the primary routes for elimination of the nerve agents studied (VX, GD, GB, GA). A recent report (de Jong *et al.*, 1991) measured the levels of GD in the kidney and urine of rats after administration of 2-6 LD_{50}s to anesthetized, atropinized, and artificially ventilated animals. It was concluded that although only 1% of the administered dose was excreted by the urinary route, this amount may produce toxicologically significant effects. However, it had been reported previously for two other species (guinea pig and marmoset) that renal excretion of intact and toxic soman isomers was at least 2 orders of magnitude lower than that identified in rats.

3.3.4.6 Conclusions

Of the nerve agents and HD considered under the worst-case scenario, VX could pose a significant hazard to handlers due to its possible persistence within the body. In addition, the possible existence of VX depots in the skin even after decontamination needs to be considered. Moreover, the high toxicity of the two isomers of soman [C(+)P(-) and C(-)P(-)] and their very slow hydrolysis by phosphonylphosphatases cause some concern, to a much smaller degree, as a possible hazard to handlers.

3.3.4.7 Data Gaps

Analysis of the fate in the body of the threat agents, especially for the worst-case scenarios defined in this report, can be expected to have numerous data gaps. Data gaps found to have a pervasive influence on preparing Section 3.3 are as follows:

- The human metabolism of VX, particularly regarding suggested enzymatic oxidation reactions, requires clarification.
- The question of agent depots needs research. If depots are positively identified, they should be characterized.
- The elimination of nerve agents from the human body appears to have been poorly studied.

3.4 POTENTIAL HAZARD OF THE DECONTAMINATED REMAINS TO HANDLERS

A potential hazard to handlers of the decontaminated remains could exist due to any residual agent in the body that was inaccessible to the surface decontaminant and was not fully detoxified in tissues and body fluids. Estimates were presented of initial maximum levels of contamination which could have been generated by three battlefield exposure scenarios: exposure to agent delivered by explosive munitions, immersion of the remains in a pool of agent, and exposure of wounds to agent. These estimates were then used to determine the percutaneous absorption of agent into the body (in toxicity units). Section 3.2.2 and Table 3.1 analyze the peak levels of contamination and absorption due to release of agents from a variety of battlefield munitions. Section 3.2.3 and Table 3.2 analyze the potential contamination of the remains from whole-body immersion in agent. Section 3.2.4 and Table 3.3 analyze the effects of wounds on contamination levels.

The extent of the eventual hazard will depend on the type of agent, its concentration, its rate of detoxification and/or dilution in body fluids, and the type of hazard it poses (contact and/or vapor). Of greatest importance is the ability to detect agents. The proposed detection devices will likely detect agent vapors with sufficient sensitivity to ensure the completeness of the decontamination process and to preclude vapor exposures from agent present in the remains. The detection devices, however, may not detect agent that is dissolved in body fluids. The presence of undetected agent would raise the possibility of a contact hazard. Contact hazard is an especially great concern with the low-volatility agent, VX, which does not generate significant levels of agent vapors. On the other extreme are high-volatility agents, such as GB, which, even in the dissolved form, would generate sufficient vapor levels to be detectable by agent monitors. Hazards due to the residual intermediate-volatility agents HD, GA, and GD must be carefully considered. While residual HD is expected to be completely detoxified in the body shortly after decontamination, the completeness of detoxification of the G agents in the body is still in question, because of the possible existence of soman depots (unlikely in man) and the generally lower affinity of hydrolytic phosphonylphosphatases for the toxic isomers of G agents (see Section 3.3).

Moreover, the extent of volatility and off-gassing of intermediate-volatility agents from solution in biological tissues and fluids is not known; vapors from these sources may be below the vapor detection sensitivity. Thus, these types of contamination of the remains could give rise to a slow, cumulative vapor and/or contact hazard to handlers. Studies are needed on the sensitivity of current methods to detect dissolved or "off-gassed" agent, especially with contaminated human tissues.

Estimates were made of levels of active agent that might be present in different body compartments of the remains and might pose a hazard to handlers who come into contact with contaminated tissues and body fluids. The following strategy was used: The exposure levels and associated percutaneous LD_{50}s determined for battlefield-munitions scenarios (see Table 3.1) and for whole-body-immersion scenarios (see Table 3.2) were used to calculate the amount of agent (in grams per casualty) that was absorbed if the same number of percutaneous LD_{50}s were administered intravenously or intramuscularly. The conversion of percutaneous toxicities to intravenous or intramuscular toxicities is predicated on the assumption that, regardless of the route of administration, the lethal effects of agents are ultimately mediated by the entry of active agent into

the bloodstream whence it is carried to and reacts with specific targets in the body (e.g., peripheral and central nervous system in the case of nerve agents). The results of these calculations are depicted in Table 3.7. The data in Table 3.7 indicate that much larger amounts of VX and TVX than of GD, GB, or TGD could remain in the bodies of casualties. For explosive munitions, the amounts of VX or TVX by either the intravenous or the intramuscular route were estimated to range from 2 to 80 times the amounts for GD, GB, or TGD. For the whole-body-immersion scenarios, the amount of VX or TVX that could remain in the casualty by either parenteral route was estimated to range from 90 to 270 times those for GD, GB, or TGD.

The data in Table 3.7 also show that the amount of agent calculated to enter the casualty is only slightly higher if the estimate is based on the intravenous LD_{50} as compared to the intramuscular LD_{50}. This correspondence reflects the ready entry of active agent (i.e., without extensive detoxification) into the bloodstream from intramuscular sites. In remains without a viable circulation, however, higher levels of agent can be expected to persist in both skin and muscle tissue and in interstitial fluids. Agent in these sites would be subject only to dispersion by diffusion and the inherent rates of detoxification and hydrolysis of agents.

The levels of active agents in skin are more difficult to estimate than are those derived from the intramuscular LD_{50}. Among the agents considered, a very limited amount of relevant data are available, for VX only, for estimating the amount of active agent (g/man) that may be sequestered in the skin of remains. Using a percutaneous-to-intravenous dose ratio of 20:1 (volar forearm) for 70% inhibition of blood cholinesterase (which produces signs of poisoning) (Sim, 1962), 4.4 g (20 X 0.22 g/casualty) and 242 g (20 X 12.1 g/casualty) of VX could be present in skin tissue in the explosive-munitions and whole-body-immersion scenarios, respectively. This analysis suggests that, theoretically, all of the available agent can be absorbed.

Table 3.7. Estimate of potential agent levels in decontaminated remains.

Agent	Maximum Percutaneous[a] Exposure Levels (g/man)	(LD_{50}/man)	Agent Remaining in Victim[b] IV[c] (g/man)	IM[d] (g/man)
		Explosive Munitions		
VX	4	400	0.22	0.34
GD	20	57	0.0068	0.013
GB	200	118	0.12	—[g]
TVX	6	960	0.54	0.82
TGD	200	570[f]	0.068	0.13
HD	200	67	—[g]	—[g]
		Whole-Body Immersion[b]		
VX	216	21,600	12.1	18.4
GD	204	592	0.071	0.13
GB	220	130	0.13	—[g]
TVX	216	34,560[e]	19.4	29.4
TGD	204	592[f]	0.071	0.13
HD	254	84	—[g]	—[g]

[a]Source: Tables 3.1 and 3.2.
[b]The maximal amounts of agents persisting in the remains was calculated by converting the percutaneous LD_{50} values to grams/casualty if one assumes that the agent had been administered either intravenously or intramuscularly. Amount of agent in remains = (total number of percutaneous LD_{50}) (i.v. LD_{50}).
[c]IV LD_{50}s for VX, GD, and GB are 0.56, 0.12, and 1.0 mg/man, respectively (Anderson, 1974; Facklam, 1982).
[d]IM LD_{50}s for VX and GD are 0.85 and 0.22 mg/man, respectively (Anderson, 1974; Facklam, 1982).
[e]TVX is 1.6 times more toxic than neat VX (Tomlinson and Samuel, 1980).
[f]The estimated toxicities for TGD employed the toxicity data for GD (see footnotes b, c, and d) and thus may be underestimated.
[g]LD_{50} values not available.
[h]Assumes residual agent film, 0.1 mm thick, covering a skin area of 2 m^2. Total volume = 200 cm^3. g/Man by whole-body immersion = 200 cm^3 x density of agent.

Actual skin levels are expected to be less than these estimated amounts but greater than the amounts calculated from the intravenous toxicities.

The overall results in Table 3.7 indicate that much larger amounts of VX than of G agents may be absorbed in the skin, muscle tissues, and body fluids of remains and may potentially pose a contact hazard to handlers.

Spontaneous and enzymatic hydrolysis are major routes of detoxification of many agents in body tissues and fluids. Irreversible binding of agents to body constituents is probably not a significant detoxifyng mechanism since, except for HD, both the specific and nonspecific biological sites would be saturated at the levels of agent under consideration. Furthermore, HD will hydrolyze rapidly in the body and thus will not be a hazard for handlers. Also, as discussed in Section 3.3 and shown in Table 3.6, G agents, compared to VX, have a greater than fivefold higher rate of spontaneous hydrolysis in humans and in numerous animal species. In addition, G agents are even more rapidly hydrolyzed by phosphonylphosphatases, whereas VX is not susceptible to enzymatic detoxification in humans. (Grotta *et al.*, 1983, Harris *et al.*, 1984, O'Brien, 1960).

The hydrolytic half-lives of G agents in suspensions of guinea pig skin are shown in Table 3.8. Also included in the table is the half-life for spontaneous hydrolysis of VX in water at pH7—probably the only means of VX detoxification in humans (see Section 3.3). Almost complete hydrolysis of G agents (i.e., 10 half-lives) is accomplished in 1-3 days in heated extracts (spontaneous hydrolysis), whereas 0.3-0.9 day is required for similar extents of hydrolysis with fresh extracts (enzymatic plus spontaneous hydrolysis). In contrast, about 17 days (10 half-lives) is required for almost complete VX hydrolysis. The rapid reduction of G-agent toxicity compared to the much slower loss of VX toxicity can be discerned even in contaminated-wound scenarios. Large amounts of agent can be deposited and absorbed into the body when the skin barrier is bypassed (see Section 3.2.4). For example, it can be calculated that less than 1 day would be required to reduce to 1 LD_{50} the i.v. toxicities due to G-agent contamination of wounds by whole-body immersion. On the other hand, about 18 days would be required for a corresponding reduction of VX toxicity.

The results clearly identify VX as the major hazard--albeit mainly a contact hazard--in the decontaminated remains.

3.5 SUMMARY AND CONCLUSIONS

(1) Even if the surface decontamination of remains were completely successful, it is possible that active agent, inaccessible to the decontaminant, could remain sequestered in the body. Sequestered agent would pose a hazard to morticians, pathologists, or others who may come into contact with contaminated body fluids or tissues. The objectives of this section were to analyze the extent of the residual contamination of the remains and estimate its hazard to handlers.

(2) The following estimates were obtained for maximum levels of contamination of remains (g/man with 2 m^2 of skin surface) with liquid HD, GD, GB, TGD, VX, and TVX in three exposure scenarios:

- Release of agent from selected munitions: 20-100 for HD, 20-200 for GB, 2-20 for GD, 20-200 for TGD, 2-4 for VX, and 6 for TVX. Up to tenfold

Table 3.8. Hydrolysis of nerve agents in suspensions of guinea pig skin.[a]

Agent	Hydrolysis in Heated Suspensions		Hydrolysis in Fresh Suspensions[b]	
	Half-life (min)	10 Half-lives (days)[c]	Half-life (min)	10 Half-lives (days)[c]
GB	230	1.6	125	0.87
GA	405	2.8	130	0.90
GD	461	3.2	47.5	0.33
VX[d]	2400[d]	16.7	2400[d]	16.7

[a]Source: Fredriksson, 1969a.
[b]Due to enzymatic hydrolysis by phosphonylphosphatases.
[c]Time required to achieve a 1024-fold degradation of agent.
[d]VX was not included in this study. Hydrolysis half-life represents the spontaneous hydrolysis in water, pH 7. No enzymatic hydrolysis is expected.

higher contamination may be possible by dispersal of agents from a spray tank or from aerosol generators of low-flying aircraft.

- Whole-body immersion in puddles of agent (254 for HD, 230 for GB, 204 for GD, 204 for TGD, 216 for VX, and 216 for TVX).
- Entry of agent through wounds. Assuming a wound size of 100 cm^2, the exposed area will be 1/200th of that for the other scenarios (100 cm^2/ 2 m^2), and the estimated maximal agent contamination would be reduced accordingly.

The contamination of remains by agent vapors or aerosols was deemed negligible because peak concentrations cannot be sustained and respiratory and circulating functions are absent.

(3) These assumptions were made in the estimation of the maximum quantity of active agent which could be present in the remains:

- The entire bare skin of the casualty (area = 2 m^2) was exposed. The casualty either was dead at the time of exposure or died shortly afterward.
- The loss of respiration precluded any significant accumulation of agent by inhalation.
- The loss of circulation favored the accumulation of agent at or near sites of entry into the body (e.g., skin, tissue, fluids, and wounds).

(4) The actual hazard to handlers depended on initial levels of contamination, the fate of agents in the body (e.g., reduction due to dilution, diffusion, hydrolysis, detoxification) and the sensitivity of detection devices for measurement of agent vapors (resulting from desorption) or of agent dissolved in body fluids and tissues (contact hazard).

(5) A strategy employing human toxicity units (LD_{50}) was developed for estimating the level of active agent present in the remains. The rationale and advantages of this strategy compared to the use of penetration rate data for the estimation of agent levels were discussed.

(6) Estimates were made of maximal levels of active agent in remains (in percutaneous LD_{50} units) following cutaneous exposure. Values in the explosive munitions and whole-body immersion scenarios, respectively, were as follows:

- VX, 400 and 21,600
- GD, 57 and 592
- GB, 118 and 130
- TVX, 960 and 34,560
- TGD, 570 and 592
- HD, 67 and 84.

These maximal levels of percutaneously absorbed agent would be expected to be largely confined to skin tissue and to diminish in a time-dependent manner due both to hydrolysis and other routes of detoxification and to diffusion into neighboring tissues and fluids. The maximal theoretical levels of active agent in blood and muscle tissue (g/casualty) were estimated for the above scenarios by assuming that the same number of percutaneous LD_{50}s had been administered either intravenously or intramuscularly.

(7) The presence of damaged skin or wounds is expected to produce higher agent levels in remains than would exposures of intact skin. It was predicted that the largest increases due to the removal of the penetration barrier would occur with the high-volatility agent, GB, would be followed by the intermediate-volatility agents, HD, GA, and GD, and would be smallest for the low-volatility agent, VX. This expectation was borne out by calculations in which the contamination of wounds was represented by the intravenous LD_{50}. Calculations of (percutaneous LD_{50} for intact skin)/ (intravenous LD_{50} for wounds) show that the presence of a 100-cm^2 wound would increase the absorption of G agents about 2000-fold compared to about 18-fold for VX. The eventual hazard to handlers will be determined by both the detoxification rate of agents and the ability to decontaminate wounds.

(8) The fates of threat agents in the body were analyzed. The following major mechanisms for the diminution of agent toxicity were considered: reaction with specific targets, reaction with nonspecific targets, spontaneous hydrolysis, enzymatically catalyzed hydrolysis, oxidative and other modes of metabolic inactivation, and excretion. Parameters which could enhance toxicity included the accumulations of agent in depots and, due to the loss of circulation, in the skin of remains. It was concluded that G agents and VX were not substantially detoxified by reaction with either specific or nonspecific targets. These targets would be saturated rapidly by the supralethal quantities of agents that could be encountered in the exposure scenarios. On the other hand, the detoxification of HD—which has a much larger number of nonspecific targets—would be substantial.

(9) Hydrolysis plays a major role in the detoxification of all agents. However, while the half-life due to the spontaneous hydrolysis of HD is measured in minutes, that of G agents is measured in hours, and that of VX is expressed in days. Furthermore, enzymes present in tissues (including skin) and blood can increase the hydrolysis rates of G agents three- to tenfold. Neither hydrolytic nor oxidative enzymes capable of detoxifying VX have been identified in humans. In the open-wound scenarios, it would take an estimated 0.5-1 day to reduce the toxicity of G agents in remains to 1 LD_{50}. In contrast, 18 days would be needed for a corresponding reduction in toxicity due to contamination with VX.

(10) It is concluded that at moderate and warm temperatures, only contamination with VX could pose a hazard to handlers due to the possible persistence of active agent in decontaminated remains. This hazard is expected to be largely a contact hazard.

3.6 KNOWLEDGE GAPS

(1) Information is incomplete on the efficacy of hypochlorite for decontamination of chemically contaminated wounds. Simple modifications of the composition of decontaminating solutions could significantly enhance the efficacy against some agents.

(2) The rates and extents of nerve agent penetration into the skin of human remains are not established.

(3) The half-lives for the hydrolysis/ detoxification of G agents and VX in human skin are unknown. The estimated half-lives shown in this report require validation.

(4) The effect of temperature on the persistence of chemical agents in relevant tissues and body fluids is unknown. Of particular concern is the potential hazard to handlers posed by the contamination of remains with G agents at cold temperatures. Since both the spontaneous and enzymatic hydrolysis rates will decrease significantly with temperature (e.g., the spontaneous hydrolysis rate for GB decreases about fourfold for each 10°C decline in temperature), remains exposed and stored at cold or freezing temperatures could retain hazardous levels of G agents.

(5) The abilities and extents of agents GA, GB, GD, and VX to desorb (off-gas) from human skin tissue and body fluids have not been established. The potential for off-gassed agent vapors to accumulate in sealed body bags is not known.

4. IMPLICATIONS OF THE DECONTAMINATION PROCEDURE ON EMBALMING AND COSMETIC RESTORATION

4.1 INTRODUCTION

A review of the existing mortuary science, forensic science, and biomedical literature has revealed limited information that directly addresses the effects of hypochlorite solutions on human remains. To best address the implications of proposed decontamination procedures on embalming and cosmetic restoration, the literature review conducted for this section has been supplemented by direct consultation with licensed embalmers, mortuary science educators, manufacturers of embalming chemicals and supplies, and a forensic pathologist.[5]

Three uses of hypochlorite solutions on human remains were identified during the course of this review: (1) by embalmers as a germicide wash for wounded, autopsied, or infectious disease-carrying remains (Mayer and Bigelow, 1990), (2) by embalmers as a lightening agent for dark-skinned remains (M. Wells, personal communication), and (3) by forensic pathologists as a tissue-dissolving agent for partially decomposed remains (Rao and Hearn, 1988). The first two applications are directed toward ultimate preservation of soft tissue, whereas the third is directed toward complete dissolution of soft tissue.

To characterize the tissue-dissolving potential of hypochlorite solutions, additional information was sought from literature that addresses the following: (1) injuries resulting from hypochlorite exposures, (2) use of hypochlorite as a wound antiseptic, (3) use of hypochlorite as an endodontic irrigant, and (4) dermal irritancy testing with hypochlorite.

From the information obtained, it is concluded that the effects of hypochlorite decontamination on human remains will be dependent on the following variables: (1) pH of the hypochlorite solution, (2) concentration of hypochlorite, (3) duration of hypochlorite exposure, (4) volume of hypochlorite solution, (5) temperature during hypochlorite exposure, (6) decomposition status of the remains, (7) wound status of the remains, and (8) vesicant exposure status of the remains. The first five factors, which are related to the conditions of the decontamination protocol, can be controlled, while the last three, which are related to the condition of the remains at the time of retrieval, cannot.

[5]A list of the individuals contacted and their organizations can be found in Appendix C.

The proposed procedures for decontamination of human remains exposed to chemical threat agents specifies the concentration of hypochlorite (5%) and the duration of hypochlorite exposure (up to two 15-minute washes or dips). It also specifies that refrigeration (temperatures of approximately 4°C) will be available during transport. To obtain safe and efficient decontamination of all chemical threat agents, this report recommends: (1) that hypochlorite solutions be buffered to pH $\geq$ 11, (2) that a large solution volume with ultrasonic mixing be used for the wash procedure, and (3) that temperature be maintained within the range of 5°-25°C during hypochlorite exposure (see Section 2). It is predicted that the proposed decontamination procedures, when performed according to the recommendations made in this report, will have the following impact on embalming and cosmetic restoration:

(1) Dissolution of soft tissue will be promoted in remains that have started to decompose, and possibly in remains that carry deep or extensive wounds and in remains that have been exposed to vesicants (either before death or in the first 24 hours postmortem). Embalming cannot be successfully performed on remains that undergo extensive soft tissue dissolution. To minimize tissue solubilization, it is recommended that the pH of the hypochlorite solutions used for decontamination be kept as close to pH 11 as possible and that temperatures be maintained as close to 5°C as possible during decontamination and transport.

(2) The appearance of the remains will be altered due to discoloration, dehydration, and saponification that are promoted by the hypochlorite solutions used for decontamination. It is possible that hair will be lost. Successful cosmetic restoration cannot be achieved if these types of changes occur in the face or hands (i.e., sites exposed during open-casket viewings).

(3) Although hypochlorite solutions are used widely as disinfectants and germicides, the solutions being recommended for decontamination of chemical threat agents may fail to promote extensive killing of decay-promoting bacteria or pathogens because of their high pH and buffering capacity. If sufficient microbial contamination is present after hypochlorite treatment, the remains will continue to undergo decomposition, and embalmers at Mortuary Dover will have to treat chemically decontaminated remains as a potential biohazard until other germicidal procedures are employed.

(4) Although hypochlorite reacts with components in embalming fluids to produce acutely toxic and

carcinogenic products, these reactions are not likely to occur under the conditions employed at Mortuary Dover and at most licensed funeral homes.

The reasons underlying these predictions are discussed in the sections below.

4.2 DISSOLUTION OF SOFT TISSUE

Hypochlorite dissolves soft tissue by a rapid exothermic reaction that results in nearly complete degradation of protein with concomitant neutralization of the hypochlorite (Baker, 1947, as reported in Hoy, 1981). According to Rao and Hearn (1988), there is no published information in the forensic science literature on the optimal conditions for hypochlorite-induced skeletonization of human remains. Removal of soft tissue from bone can be accomplished by boiling bones in Clorox (5.25% sodium hypochlorite at pH 10.8-11.4) or by immersing bones in swimming pool chlorine (10% sodium hypochlorite in 3.5M sodium hydroxide at pH 13.2-13.5) at room temperature (W. Hearn, personal communication).

The rapid action of pool chlorine on soft tissue was demonstrated during an accident in which the victim was pinned under an overturned tanker that spilled hypochlorite at the rate of 5 gal/min on his lower body (Rao and Hearn, 1988). The victim, who died within 10 minutes of hypochlorite exposure, sustained erosion of soft tissue to the level of bone while still alive. The soft tissue erosion continued postmortem, despite dilution of the chemical spill with large volumes of water. Skin sites on parts of the victim's body that had minimal contact with hypochlorite displayed pitting. (Photographs depicting the soft tissue erosion and skin pitting can be found in the publication.) Progressive soft tissue erosion was also seen in an endodontic patient who was inadvertently injected in the cheek with 0.5 ml of 5.25% sodium hypochlorite that was analyzed to be at pH 12.9 (Gatot *et al.*, 1991). In this case, the necrotic injury also began at the time of hypochlorite exposure, but it was arrested three days later by surgical intervention. A controlled study in rabbits revealed that a hemorrhagic reaction occurs immediately following intradermal injection of undiluted Clorox (Pashley *et al.*, 1985). In a study in guinea pigs, however, subcutaneous implantation of polyethylene tubes filled with pH-12 sodium hypochlorite at concentrations of 0.9-8.4% produced no observable tissue damage relative to saline-filled control tubes (Thé *et al.*, 1980).

There are no reports of severe tissue-dissolving injuries occurring with lower-pH or lower-concentration hypochlorite solutions. Furthermore, it is not known whether the same hypochlorite solution that caused tissue dissolution after inadvertent injection in the endodontic patient would cause a similar type of injury after topical exposure. Undiluted Clorox does not cause acute necrotic injuries after dermal exposure. Nixon *et al.* (1975) found that severe irritation but no tissue destruction was produced in the intact skin of healthy volunteers (n=7) after exposure to 1 ml of 5.25% hypochlorite bleach in a 4-hour patch-test protocol. Four of the seven subjects in this study displayed weeping and eschar reactions in addition to erythema and edema at the test sites. These results are in contrast with the results of more recent studies conducted by Maibach (reported by Gum, 1991; Wanat, 1991a; Wanat, 1991b). In these studies, healthy volunteers (n=20) displayed no skin irritation 0.5, 2, or 24 hours after application of sodium or calcium hypochlorite at concentrations of 0.5, 2.5, or 5.0%. However, mild irritation was observed at the 24-hour time point in five of ten subjects who had test solutions reapplied to the same exposure sites 2 hours after the initial application. In a recent patch-test study, the irritant properties of 1% sodium hypochlorite made to different pHs with sodium hydroxide were determined in healthy volunteers. The extent of dermal irritation was found to be nonlinearly dependent on solution pH over the range of 11.17-13.06 (Hostynek *et al.*, 1990). Of key importance in this study was the buffering capacity of skin. The most extensive irritation was obtained when 100 µl of a pH-12.19 hypochlorite solution was applied; however, at the end of the 24-hour exposure period, the mean pH of the retrieved solution was measured to be 8.11. The test solution with the highest initial pH—13.06—was lowered to pH 9.40 at the end of the exposure period.

A recent study at the Battelle Medical Research and Evaluation Facility compared the extent of irritation produced by 0.5% and 5.0% sodium hypochlorite solutions on intact and abraded rabbit skin (reported by Gum, 1991). Rabbits exposed to the 0.5% solution had a mean primary dermal irritation index (PDII) score of 0, whereas those exposed to the 5.0% solution had a PDII score of 2.6. The PDII score incorporates ratings for both intact and abraded skin. Calcium hypochlorite was found to be more irritating than sodium hypochlorite, inasmuch as it produced a PDII score of 0.3 at the 0.5% concentration. Nixon *et al.* (1975) reported PDII scores of 1.2 and 0.8 for rabbits and guinea pigs, respectively, that were exposed to 5.25% hypochlorite bleach. Of interest is their finding that humans display a more intense irritation reaction than either test animal when similar exposure conditions are used on intact skin.

Based on the available literature and toxicology reports (Cullen and Taylor, 1918; Nixon *et al.*, 1975; Gosselin *et al.*, 1984, as reported in TOXNET, date

unknown; Bloomfield and Sizer, 1985; Hess *et al.*, 1991, Rumack and Spoerke, 1991, as reported in TOXNET, date unknown), it appears that the dermal injuries produced by exposure to hypochlorite solutions (concentration ≤ 5.25%, pH ≤ 11.4, volume is ≤ 1 ml, and incubation time ≤ 24 hours) are inflammatory in nature, and that sometimes there is involvement of an allergic or hypersensitization response. Because a functioning circulatory system is required for development of these injuries, they are not considered relevant for prediction of hypochlorite effects on human remains. The injuries that are considered directly relevant to postmortem tissue dissolution are those that are manifested immediately after exposure and involve destruction of tissues via rapid direct chemical reaction mechanisms.

A key question that cannot be answered from the available literature on *in vivo* hypochlorite effects is whether there are definitive thresholds (or combination thresholds) in concentration, pH, exposure time, temperature, and solution volume for production of acute necrotic injuries. In this regard, Mack (1983) states that 12.5 is the critical pH for production of esophageal ulcers by ingested hypochlorite solutions. However, in controlled studies in dogs, Yarington (1970) was able to produce caustic esophageal lesions with Clorox by using large solution volumes (10 ml) and long exposure times (10 minutes). Yarington claims that relatively few serious esophageal injuries are seen after accidental ingestion of Clorox because the solution volumes are low (usually one swallow) and the exposure times are short (the victim vomits up the solution and early medical intervention includes dilution with water or a neutralizing acidic solution).

A number of controlled studies have examined the tissue-solubilizing properties of hypochlorite solutions *in vitro*. The earliest study was conducted in the World War I era, when buffered 0.5% hypochlorite was extensively used as a wound antiseptic (Taylor and Austin, 1918). The goal of this study was to identify the optimal conditions for chemical debridement of necrotic wound tissue. More recent investigations have focused on identifying the optimal hypochlorite solution for removal of necrotic pulp and debris during surgical preparation of the root canal (Grossman and Meiman, 1941; Trepagnier *et al.*, 1977; Hand *et al.*, 1978; Thé, 1979; Cunningham and Balekjian, 1980; Thé *et al.*, 1980; Abou-Rass and Oglesby, 1981; Gordon *et al.*, 1981; Liu *et al.*, 1993; Nakamura *et al.*, 1985; Pashley *et al.*, 1985). Various *in vitro* tissue preparations that derive from dental pulp, connective tissue, skin, uterus, or liver have been incubated with hypochlorite in the concentration range of 0.5-10%. Tissue preparations that are freshly isolated or quickly frozen and thawed have been used as models of vital tissue, whereas those that are left at ambient or elevated temperatures for extended periods have been used as models of necrotic tissue. The extent of tissue dissolution over time has been determined by measuring tissue weights, sediment volumes, or amounts of hydroxyproline released into the hypochlorite solution. Alternatively, some investigators have reported the time to complete tissue dissolution as determined by visual inspection.

The combined results of the *in vitro* tissue dissolution studies suggest that dissolution of necrotic tissue is more rapid than dissolution of vital tissue (Basher, 1917, as reported in Taylor and Austin, 1918; Gordon *et al.*, 1981)[6] and that dissolution of tissue is dependent on (1) hypochlorite concentration (Taylor and Austin, 1918; Trepagnier *et al.*, 1977; Hand *et al.*, 1978; Thé, 1979; Abou-Rass and Oglesby, 1981; Gordon *et al.*, 1981; Liu *et al.*, 1983; Liu *et al.*, 1993; Nakamura, *et al.*, 1985), (2) solution pH (Taylor and Austin, 1918), (3) incubation time (Trepagnier *et al.*, 1977; Thé, 1979; Gordon *et al.*, 1981; Nakamura *et al.*, 1985), (4) incubation temperature (Cunningham and Balekjian, 1980; Abou-Rass and Oglesby, 1981; Nakamura *et al.*, 1985), and (5) solution volume (Thé, 1979; Gordon *et al.*, 1981).

A 72% mean reduction in tissue weight was detected by Hand and co-workers (1978) when necrotic skin and subcutaneous tissue from rats was incubated in 5.25% sodium hypochlorite at 10 mg tissue/ml of solution for 7 minutes at room temperature. Essentially no tissue was dissolved, however, when the hypochlorite concentration was reduced to 0.5% and the same incubation conditions were maintained. Thé (1979) found that the amount of necrotic rat abdominal wall tissue solubilized by 3% hypochlorite in 20 minutes was 2.3-fold greater than that solubilized by 1% hypochlorite (70% versus 30% of starting tissue weight). However, Gordon *et al.* (1981) reported that 1%, 3%, and 5% hypochlorite were equally effective at solubilizing necrotic dental pulp (90% dissolution in 10 minutes). Taylor and Austin (1918) demonstrated that 0.5% hypochlorite solutions that were strongly alkaline (made to 0.1 N sodium hydroxide) were more effective at solubilizing macerated rabbit or cat liver than weakly alkaline solutions at the same concentration (Dakin's solution, which is buffered to pH 9 with boric acid). Although it was found that strongly alkaline solutions were able to dissolve tissue at hypochlorite concentrations as low as 0.1%, weakly alkaline solutions lost their ability to solubilize tissue when the hypochlorite concentration was ≤ 0.2%.

[6] In contrast to the results of other investigators, the results of Abou-Rass and Oglesby (1981) suggest that fresh tissue is dissolved more rapidly than necrotic tissue.

The outermost layer of the skin epidermis–the stratum corneum–is an acellular layer composed of interlocking keratin protein fibers, lipid, and water. Because the epidermis resists penetration of exogenous substances, there is reason to believe that topologically intact skin (i.e., skin that is closed and oriented in the same direction as it is *in situ*) is more resistant than other tissues to hypochlorite-induced dissolution. This concept is supported by Duffy *et al.* (1986), who state that hypochlorite solutions are unable to dissolve necrotic skin and that the outer layer of skin should be removed before treating burns with hypochlorite. It is therefore predicted that remains that are retrieved in good physical condition shortly after death will not succumb to rapid soft tissue erosion when the proposed decontamination procedures are employed, provided that the pH of the hypochlorite solution is maintained close to 11 and that the temperature is maintained close to 5°C during decontamination and transport.

During decomposition of human remains (see Section 4.4 for a general discussion of decomposition), there is separation of the outer layer of the skin–the epidermis–from the underlying dermis (Mayer and Bigelow, 1990; Strub and Darko, 1989). This desquamation reaction, which is termed "skin slip," can manifest itself in two ways. If a significant amount of internal decomposition has taken place, the generated tissue gases will force purge or other body fluids into the dermal-epidermal clefts to form large (7.5- to 15-cm diameter), foul-smelling blisters. If there is no expulsion of fluid into the cleft, the skin will appear intact, but overt separation of the epidermis from the dermis will occur during mechanical handling of the remains.[7] (This effect is analogous to Nicholsky's sign in patients with bullous diseases.) It is predicted that areas of skin slip on more extensively decomposed remains will provide an entryway for hypochlorite penetration into the underlying soft tissues. The decomposed soft tissue of the human remains will be rapidly dissolved by hypochlorite in the same manner as necrotic soft tissue in wounds.

Like skin slip, wounds provide a direct entryway for reactive hypochlorite into the soft tissues. However, if remains with wounds are retrieved very shortly after death,[8] only localized areas of decomposition around wound sites should be subject to rapid dissolution.

[7]The mechanical aspects of the decontamination procedure–ultrasonic mixing of the decontaminating hypochlorite solutions (see Section 2) or high-pressure washing with hypochlorite and/or water–will probably promote dermal-epidermal separation in remains that have begun to decompose.

[8]The onset of body-wide decomposition is earlier in remains that carry wounds or unhealed surgical incisions (Mann *et al.*, 1990); Mayer and Bigelow, 1990).

Exposure to vesicants such as HD produces pathogenic changes at the dermal-epidermal junction of living individuals (Papirmeister *et al.*, 1991) that are similar to those of skin slip. For this reason, it can be expected that the remains of individuals exposed to vesicants will be particularly prone to severe desquamation over time and during handling. This effect is likely to occur even if death took place prior to the development of visible HD lesions or to HD exposure, since microscopic dermal-epidermal separations are produced shortly after HD exposure in excised or organ-cultured human skin samples (Moore *et al.*, 1986; Mol *et al.*, 1991). Because remains exposed to vesicants will have an entryway for hypochlorite penetration into soft tissues, they may be subject to tissue dissolution. However, the solubilization effect, if it occurs, would be less pronounced than that for remains that have begun to undergo decomposition.

4.3 ALTERATIONS IN APPEARANCE

There is much controversy regarding the use of hypochlorite solutions in the funeral industry (R. Bradford, personal communication; D. Flowers, personal communication; C. Robinson, personal communication; Sawyer, 1987). The factors influencing the controversy include: (1) a recommendation by the Centers for Disease Control (CDC) that hypochlorite be used as the universal disinfectant for human immunodeficiency virus (HIV), hepatitis B virus (HBV) and other blood-borne pathogens in health-care settings (CDC, 1987, 1988, cited in Mayer and Bigelow, 1990); (2) statements such as "It is advantageous to routinely use sodium hypochlorite in the washing of ALL bodies," which appear in a recently published textbook on embalming (Mayer and Bigelow, 1990); (3) promotional materials from the manufacturers of embalming chemicals indicating that embalming solutions having high formalin concentrations are completely effective at destroying HIV, HBV, tuberculosis bacilli, fungi, and all other infectious microorganisms (Sawyer, 1987; C. Robinson, personal communication; M. Wells, personal communication); and (4) warnings from professional organizations and from state and local licensing authorities indicating that hypochlorite reacts with formaldehyde to produce toxic and carcinogenic gases (National Funeral Director's Association, 1988).

No literature addressing the cosmetic effects of hypochlorite on human remains was identified. Embalmers interviewed for this report indicated that the presence of hypochlorite at 1000-5000 ppm (0.1-0.5%; the concentration recommended for disinfection and germicidal purposes [Mayer and Bigelow, 1990])

on human remains for extended periods of time (i.e., several hours to days) does not prevent successful restoration of the face and hands for casket-viewing purposes (R. Walker, personal communication; E.C. Ogrodnik, personal communication). Although this procedure is contrary to recommended safe embalming practice, low concentrations of hypochlorite have been mixed with arterial embalming fluid to lighten the remains of dark-skinned individuals (M. Wells, personal communication).

There is presently no recommended use in the funeral industry for 5% hypochlorite or undiluted Clorox on human remains. However, there are extensive rumors of its indiscriminate use on autopsied or infectious-disease-carrying remains (D. Flowers, personal communication). In these cases, open-casket viewing was ruled out due to the public health concerns associated with the original disease state. However, it is claimed that copious use of undiluted Clorox produced color changes in the skin, hair, and nails that could not be corrected by cosmetics. (Hair is rumored to have turned bright green.)

Two additional cosmetic problems associated with the use of 5% sodium hypochlorite have been raised by individuals interviewed for this report: (1) Dehydration (i.e., mummification) of the remains due to the osmotic pressure of the external hypochlorite solution (E.C. Ogrodnik, personal communication); and (2) saponification of the body fat in a manner analogous to that seen in the poultry industry following use of sodium hypochlorite dip solutions at high pH[9] (R. Bradford, personal communication). Both dehydration and saponification would alter the appearance of the skin: Dehydration results in browning (which will not occur in hypochlorite) and a leatherlike texture, while saponification results in an unnatural, waxy appearance (Mayer and Bigelow, 1990; Strub and Darko, 1989). Although these changes could prelude acceptable cosmetic restoration, bacterial decomposition of the affected tissues would be retarded.

It has been reported that individuals exposed to vapors from alkaline hypochlorite cleaning solutions undergo acute reversible hair loss (Stuttgen, 1991, as reported in TOXNET, date unknown). Signs of hair dystrophy and loss of the hair sheath have been associated with the toxic alopecia. Histological characterization has demonstrated infiltration of lymphocytes into affected skin areas. If an inflammatory response is a prerequisite for hypochlorite-induced hair loss, then this effect is not relevant to the discussion of hypochlorite effects on human remains. Furthermore, the remains of hairless individuals (e.g., cancer patients that received cytotoxic chemotherapy) can be restored cosmetically to an acceptable appearance with the aid of wigs, eyebrows, moustaches, and beards.

[9]Saponification, which results in the production of soap, is the hydrolysis of fat by alkali.

4.4 ATTENUATION OF GERMICIDAL ACTIVITY

The germicidal actions of hypochlorite and other chlorine-containing compounds against viruses, bacteria, and fungi have been well characterized since their introduction as disinfectants in the mid-1800s (Sykes, 1967; Dychdala, 1991). The effectiveness of hypochlorite as a germicide is highly dependent on pH, concentration, temperature, and amount of contaminating organic material present. Maximal activity is obtained with low pH, high hypochlorite concentration, high temperature, and no competing protein source. Solution pH is particularly important because hypochlorous acid, which is favored at neutral pH, is approximately two orders of magnitude more effective as a bacteriocidal agent than hypochlorite anion, which is favored at alkaline pH. A similar pH dependency has been shown for virucidal and fungicidal activity. As discussed in Section 2, however, hypochlorite anion is the species needed for safe and efficient decontamination of the chemical threat agents. At pHs of 11 or greater, which are recommended for chemical decontamination, all available hypochlorite is in the less bacteriocidal, anionic form. (Figure 2.1 shows the effect of pH on the equilibrium between hydrochlorous acid and hypochlorite anion.)

Undiluted Clorox, which has a measured pH of 10.8-11.4 straight from the container, is widely regarded as an effective germicidal agent (CDC, 1987, 1988, cited in Mayer and Bigelow, 1990; W. Hearn, personal communication). One factor that is probably key to the germicidal activity of this product is the absence of buffering. Downward fluctuations in the pH of unbuffered hypochlorite solutions can result from contact with acidic or neutral substances (including living tissue and physiological fluids, which are highly buffered) or from dilution. Because it is recommended that the hypochlorite solutions used for decontamination of chemical threat agents be tightly buffered to pH $\geq$ 11, it is possible that germicidal activity will be significantly attenuated relative to that of unbuffered hypochlorite solutions that are used under otherwise identical conditions. The practical implications of attenuated germicidal activity are continued decomposition of remains and possible pathogenic spread.

Decomposition of remains occurs by two general mechanisms—autolysis of cells by released lysosomal

enzymes and bacterial decay—with the latter mechanism playing a more prominent role than the former (Mayer and Bigelow, 1990; Strub and Darko, 1989). (A third mechanism, digestion by insects or other carrion-consuming fauna, becomes prominent when untreated remains are exposed for long periods of time.) Both decomposition and the spread of pathogens can be arrested by impregnating remains with formalin-containing embalming solutions (Sawyer, 1987; Mayer and Bigelow, 1990; Strub and Darko, 1989). These solutions, which promote the formation of cross-links in cellular and extracellular protein components, inactivate lysosomal enzymes and kill decay- or disease-promoting bacteria. In the funeral industry, it is routine to initiate germicidal and preservation processes at the time a body is retrieved by coating surface tissues with a formalin-containing spray and by including embalming powder in the body bag (Mayer and Bigelow, 1990; Strub and Darko, 1989). In addition, open wounds, which are a source of pathogenic bacteria, are sealed by painting on formalin-containing autopsy gel. Because formalin (aqueous formaldehyde) is not compatible with hypochlorite under some conditions (see Section 4.5 for additional details), tissue-preserving procedures cannot be initiated on chemically decontaminated remains until they are washed free of hypochlorite. In the absence of hypochlorite-induced germicidal activity or surface preservation, the only mechanism that will retard decomposition or pathogenic spread is refrigeration, which is expected to be available from APOE onward.

The extent of decomposition that occurs in chemically decontaminated remains in the hours between retrieval of remains and shipment to CONUS will be dependent on the following factors: (1) time since death, (2) temperature, (3) the condition of the body at the time of death, and (4) the extent of decomposition that occurred prior to body retrieval. Factors that promote decomposition include heat and the presence of diseases and/or injuries (Mann *et al.*, 1990; Mayer and Bigelow, 1990; Strub and Darko, 1989). Although decomposition does not generally begin until approximately 36 hours after death, environmental conditions and disease state can influence the process so that it begins within several hours postmortem (Mann *et al.*, 1990; W. Hearn, personal communication). A long-term survey of the effects of various factors on the decomposition of human remains is under way at the University of Tennessee, Knoxville (Mann *et al.*, 1990). Retardation of decomposition is crucial when the proposed decontamination procedure is employed, since tissue dissolution is predicted to occur when decomposed remains are exposed to hypochlorite (see Section 4.2).

Although refrigeration of unembalmed remains delays the onset and slows the rate of decomposition, it promotes two forms of skin discoloration: livor mortis (which is intravascular) and postmortem stain (which is in the tissues). Remains that are exposed to cold for long periods tend to turn orange and then brown (Mann *et al.*, 1990).

The potential health hazards associated with continued growth of pathogenic bacteria during decontamination and shipment of remains are obvious. In routine funeral industry practice, decomposition of remains presents two major problems to the embalmer: (1) intense, nausea-provoking smells; and (2) the inability to achieve successful preservation and/or cosmetic restoration because of skin slip (see Section 4.2 for a discussion of skin slip). Even in the absence of rapid, hypochlorite-induced soft tissue dissolution, skin slip can be a problem. Prior to arterial embalming, blisters must be drained and any epidermis that has separated from the dermis must be removed (Sawyer, 1992; Mayer and Bigelow, 1990; Strub and Darko, 1989). All resulting "blister beds" need to be sealed with autopsy gel to prevent outflow of embalming fluids during high-pressure injection procedures. If large areas of skin are lost to skin slip, successful embalming cannot be achieved. In addition, cosmetic restoration cannot be performed when extensive skin slip has occurred on the face.

4.5 REACTIONS WITH EMBALMING CHEMICALS

Funeral industry publications warn embalmers of the hazardous substances that can be produced when hypochlorite reacts with compounds contained in embalming fluids (Douthit, 1992; M. Wells, personal communication). The most noted of the reactions occurs between hypochlorite and formaldehyde. Formaldehyde is ubiquitous in the embalmer's preparation room, since it is released from all embalming fluids, sprays, and gels that contain formalin. Under certain conditions, hypochlorite decomposes to yield molecular chlorine, which reacts with formaldehyde to liberate hydrogen chloride gas and phosgene (a pulmonary irritant). If methanol is present, hydrogen chloride reacts with formaldehyde to form chloromethyl methyl ether, a recognized carcinogen (Chloromethyl methyl ether, 1992).

$$\underset{\text{chlorine}}{2Cl_2} + \underset{\text{formaldehyde}}{CH_2O} \longrightarrow \underset{\text{phosgene}}{COCl_2} + \underset{\text{hydrogen chloride}}{2HCl}$$

$$HCl + CH_2O + CH_3OH \rightarrow ClCH_2OCH_3 + H_2O$$

hydrogen chloride; formaldehyde; methanol; BCME; water

The conditions required for production of phosgene or BCME have been characterized. The likelihood of producing these hazardous gases when embalming is performed on remains that have been decontaminated with hypochlorite is low for two reasons: (1) Both reactions require free chlorine, which is not available at the high pHs recommended for safe and efficient chemical decontamination, and (2) both reactions require direct sunlight (Booth, 1985).

A third possible reaction between molecular chlorine and formaldehyde, which results in the production of toxic carbon monoxide gas, is also unlikely to occur because it requires heat.

$$Cl_2 + CH_2O \rightarrow CO + 2HCl$$

chlorine; formaldehyde; carbon monoxide; hydrogen chloride

According to the National Funeral Directors Association (1988), the following compounds that are used in the embalmer's preparation room can also potentially react with oxidizers or halogenated compounds:

- Dimethylformamide
- Ethylene glycol
- Glutaraldehyde
- Hexylene glycol (component of arterial embalming fluid)
- Isobutane (component of deodorizing sprays and cosmetics)
- Isopropanol
- Naphtha
- ortho-Dichlorobenzene
- para-Dichlorobenzene
- Paraformaldehyde
- Phenol
- Propane

Based on a review of the chemical literature, it is unlikely that any of the possible undesirable reactions would take place under the conditions prevailing at Mortuary Dover.

5. SUMMARY

This review of the literature addressed issues specific to the decontamination of chemically contaminated remains with emphasis on three questions: (1) Does the decontaminant work? (2) Even after decontamination, is a residual level of agent present, i.e., in depots or pools, that could pose a hazard to someone handling the remains without adequate chemical agent protection? (3) Are any cosmetic effects produced by the decontamination procedure(s)? These questions, and others, are important because the Army is preparing doctrine for a specific field system for the decontamination of chemically contaminated remains. It is important that the decontamination procedures under development allow for the release of the remains for funeral rites as desired by the family of the deceased.

Three exposure scenarios were considered in our analysis: (1) immersion of the remains in a puddle of agent, (2) agents delivered in munitions, and (3) remains with wounds. Associated factors that were incorporated into our analysis included the amount of agent to be decontaminated, the area of coverage, the volume of the decontamination tank, the time required for contamination, and the extent of wounds. The agents considered were GA, GB, GD, VX, HD, TGD, and TVX. For the three scenarios listed above, these conclusions were reached:

(1) Efficacy of decontamination. The information at hand suggests that a hypochlorite solution that would be an optimal decontaminant for all agents under all conditions is not currently available. A compromise between the optimal conditions for decontamination of each agent can be made to produce conditions that are adequate for decontamination of all agents. Based on available rate data for the hydrolysis of the agents and information on toxic products resulting during decontamination of agents, a limited range of solution conditions is suitable for adequately decontaminating all of the agents considered in this report:

- Alkaline conditions (pH of 11 to 12)
- Hypochlorite solutions > 5.0%
- Time of immersion in decontamination solution to be determined by temperature of the bath.

It is not clear whether the use of hypochlorite solution in the proposed procedure will be adequate for the decontamination of thickened agents (TVX and TGD). Thickener(s) might make decontamination slower and more difficult by preventing or limiting access of the decontaminant to the agent. On the other hand, the small amount of thickener in thickened agents might have little or no effect on the availability of the agent to the decontaminant, and hence little change in rate of decontamination might be seen.

By-products of agent decontamination can also be sufficiently toxic to require cautions handling, i.e., sulfoxides and sulfones produced by decontamination of HD in alkaline solutions.

(2) Possibility that residual levels of agent would persist in remains and pose a hazard to handlers without protective gear. To address this question, three exposure scenarios were considered:

- Agent(s) delivered by munitions. This is the most likely scenario for exposure of remains to agent. The potential peak contamination levels were determined using deposition/area coverage charts and dosage/area coverage charts.
- Whole-body immersion, the worst-case scenario.
- Remains having wounds. The presence of wounds was considered to be an important factor because a large amount of agent could accumulate at the wound site in the absence of a viable circulation to remove, dilute, and detoxify the agent(s) absorbed. The analysis indicates that the presence of wounds would markedly increase the absorption of GD and GB but would have much less effect on VX absorption.

For all three scenarios, the total amount of agent(s) entering the casualty was calculated (based on known LD_{50} values). Estimates were also made of the amounts of active agent that might be present in body compartments and skin. The overall results indicate that much larger amounts of VX than G agents may be absorbed in the skin, muscle tissues, and body fluids.

It was concluded that the worst-case agent was VX because of its high rate of percutaneous absorption and low rates of spontaneous and enzyme-mediated detoxification. At moderate and warm temperatures, only contamination of remains with VX should pose a hazard to handlers due to the possible persistence of active agent in decontaminated remains. This hazard is expected to be largely a contact hazard and not a vapor hazard. Whether VX persistence presents an exposure problem is not clear. (Under conditions of extreme cold, the G agents might also pose a similar hazard even after the decontamination procedure.)

(3) Potential cosmetic effects produced by decontamination procedures(s). It is quite possible that cosmetic aspects of the remains will be affected by the decontamination procedure. The effects of hypochlorite decontamination on human remains will be dependent on the following variables:

- pH of the hypochlorite solution
- Concentration of the hypochlorite solution
- Duration of hypochlorite exposure

- Volume of hypochlorite solution
- Temperature during hypochlorite exposure
- Decomposition status of the remains
- Wound status of the remains
- Vesicant exposure status of the remains.

The first five factors, which are related to the conditions of the decontamination protocol, can be controlled within certain limits, while the last three, which are related to the condition of the remains at the time of retrieval, cannot.

There is no definitive information on the extent of tissue dissolution or the alterations in appearance that will occur after a 15-30-minute exposure to 5% hypochlorite that has been buffered to pH 11 and maintained at 5°-25°C.

(4) Data/Information Gaps

Also identified in this report are the following areas in which information pertinent to the unequivocal resolution of the questions addressed was found to be limited or lacking. These data gaps included:

- The hydrolysis rate for GB in acidic solution and in alkaline solutions under field conditions
- The rate of reaction of HD with hypochlorite under both acidic and alkaline conditions
- Information on the reaction of hypochlorite with thickened agent(s)
- The efficacy of hypochlorite in decontaminating wounds
- Rates and extent of penetration of agent into the skin and surrounding tissues of human remains
- The half-lives for the hydrolysis of G agents and VX in human skin
- The effect of temperature on the persistence of chemical agents in relevant tissues and body fluids
- The extent of off-gassing of agents from human skin, tissue, and body fluids
- Definitive information on the effects of hypochlorite on skin of human remains, e.g., dissolution effects and alterations in appearance.
- Ability and practicability of buffering hypochlorite solutions to appropriate pH during repeated treatments of human remains
- The extent of depletion of hypochlorite during repeated treatments of human remains
- The existence and characterization of enzymes in human tissues for the degradation of VX
- Dissolution of human tissue at pH < 7.
- Persistence of agents in human skin and other tissues
- Effectiveness of ultrasound and other mechanical dispersal techniques in the decontamination of HD and thickened agents
- Absorption of HD from wounds
- The existence and importance of agent depots
- Elimination routes of chemical agents
- The detection of agents in human tissues and fluids, and the detection of agent off-gassing.

[Note: A bibliography of the sources cited in the report is attached, as are three appendixes containing supplemental information.]

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APPENDIX A

Conceptual Chemical and Biological Attacks[†]

[†]Selected information from: McNally, R.E., M.M. Stark, J.M. Powers, Jr., and M.A. Sanzone (1992). *Worldwide Chemical/ Biological Threat to U.S. Air Bases Vol. I: Agent Characteristics*, data taken from Appendix B. Science Applications International Corp., Joppa, Maryland.

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Conceptual Chemical Attacks

POTENTIAL THREAT SYSTEMS

The potential threat systems that have been selected for presentation have been categorized by the range of the delivery system. The shorter range systems include terror device filled with anthrax, a mortar attack using a mustard (HD) agent fill, an artillery attack with an anthrax fill and a multiple rocket launcher attack also with an anthrax fill. Most of the systems represented in this study are moderate range systems. The moderate range systems can be divided into two basic categories; aircraft delivered systems and tactical ballistic missile systems. The aircraft delivered systems include an anthrax spray attack and a number of bomb attacks filled with hydrogen cyanide (AC), sarin(GB), sarin/GF (GB/GF), mustard-lewisite (HL), mustard (HD), and thickened soman (TGD). The tactical ballistic missile attacks included submunitions filled with anthrax, sarin (GB), soman (GD), and VX and bulk filled warheads filled with sarin (GB), thickened soman (TGD), and thickened (TVX). An anthrax filled intercontinental ballistic missile was the sole representative of a long range system.

It is important to recognize that these representative systems are certainly not comprehensive but represent a significant sample of the systems that have been known to be fielded or developed. Variations in the possible characteristics chosen by any particular adversary could cover quite a potential range. The number, location, and the time of function of the different munitions constitute the "fireplan" for the attack which would depend on the concept of operations that an adversary would have in planning an attack. The range of munitions characteristics certainly include the "effective" fill weight; the ratio between liquid and vapor released; the median droplet size of liquid; the height of release; the initial cloud size; the temperature of the liquid; the agent/burst ratio; the viscosity of thickened liquids; the velocity and altitude of release for bulk release missile systems; and the orientation of the falling line of the bulk release filled warheads relative to the wind. As important as the fireplan and munition characteristics are, the weather conditions which influence the dispersion and transport of the agent after the munition functions has been found to create a wider range of potential outcomes in many cases.

Because the transport of chemical agents typically involves significant agent transport over distances ranging from less than a kilometer to several tens of kilometers, the key meteorological parameters of atmospheric stability category, temperature, and wind speed are represented as being constant. For the chemical cases, three meteorological conditions were adopted for every case. The low temperature case is typical of a night release during the winter in the temperate climatic zones. This case used a stable, or inversion atmospheric stability, Pasquill Stability Category E; a low wind speed of 1.5 meters/second; and a temperature of 4°C (40°F). The moderate temperature case is typical of dawn/dusk conditions during the spring or fall in the temperate climatic zones. This case used a neutral atmospheric stability, Pasquill Stability Category D; a moderate wind speed of 3 meters/second; and a temperature of 25°C (77°F). The third temperature case is typical of

extreme conditions which occur during the summer in areas like Southwest Asia. This case used an unstable, or lapse atmospheric stability, Pasquill Stability Category B; a high wind speed of 6 meters/second; and a temperature of 49°C (125°F).

The transport of biological agents typically involves significant agent transport over distances of tens to hundreds of kilometers. Atmospheric stability remains the single most important factor in characterizing the transport of biological agents. For each of the regional climates- Central Europe summer, South Korea winter, Southeast Asia jungle, Southwest Asia desert- a representative time of day is used to establish levels of stability category, wind speed, and agent decay rates.

Anthrax agent decay rate is sensitive to sunlight. Daylight conditions result in agent decay of approximately 1%/minute while night conditions result in decay of approximately 0.1%/minute. Anthrax survival and ability to infect is relatively insensitive to temperature changes.

A number of different charts have been prepared for each of the attacks. The characteristics of these different charts follows:

Use of Deposition Area Coverage Charts

The deposition area coverage charts show the total liquid deposition caused by an attack. Note that the graph shows the area coverage in square meters and deposition in milligrams per square meter. Both the area coverage and the deposition level are logarithmically scaled. A single line relates the area covered by at least the deposition level on the horizontal axis. Deposition area coverage curves can be used to characterize the peak deposition level to be expected on the target (at low area coverages) or the maximal extent of liquid contamination (at low deposition values). At depositions to the left of the shoulder in the curve, area coverage is relatively insensitive to changes in deposition level. However, at depositions to the right of the shoulder in the curve, area coverage is sensitive to changes in deposition level. Liquid deposition is the most important element to examine for attacks with agent VX since the impact of falling droplets account for casualty generation [and vapor levels of VX are usually not sufficient to contribute to casualty generation]. Liquid deposition levels can contribute to casualty generation for all chemical agents [minimal impact for hydrogen cyanide], but for all agents except VX, the contribution to casualty generation is NOT a dominant effect. The liquid deposition and the size of the droplets determine the rate of vaporization in a given climate on a particular surface. The rate of vaporization determines the "secondary vapor" hazard which is the principal hazard for intermediate volatility agents (such as tabun (GA), soman (GD), mustard (HD), lewisite (L), and GF). Liquid deposition levels and the area covered by liquid deposition define both the spatial and temporal hazard areas associated with a particular attack. The particular level of liquid deposition has important implications for the contamination survivability of materials and equipment exposed to direct liquid contact.

Use of Concentration Area Coverage Charts

The concentration area coverage charts show the vapor concentration at specific times following an attack. Note that the graph shows the area coverage in square meters and concentration in milligrams per cubic meter. Both the area coverage and the concentration level are logarithmically scaled. A series of lines relates the area covered by at least the concentration level on the horizontal axis at specific times after the attack. Concentration area coverage curves can be used to characterize the peak concentration level to be expected on the target (at low area coverages) or the maximal extent of vapor contamination (at low concentration values). At concentrations to the left of the shoulder in the curve, area coverage is relatively insensitive to changes in concentration level. However, at vapor levels to the right of the shoulder in the curve, area coverage is sensitive to changes in concentration level. Vapor levels for very volatile agents such as hydrogen cyanide (AC), phosgime oxime (CX), phosgene (CG), and sarin (GB) can reach very high levels very rapidly. This results in curves which extent to the right on the graphs at times close to the time of munition function. As time passes and the agent cloud diffuses, the peak levels diminish and the area coverage increases.

Vapor levels for intermediate volatile agents such as tabun (GA), soman (GD), mustard (HD), lewisite (L), and GF tend to generate vapor concentration levels of more moderate levels (compared to the volatile agents) over a longer period of time since it may take several hours for complete agent evaporation. Increases in area coverage over time may not be as wide as for volatile agents because much lower levels of concentration are achieved. The alongwind and crosswind distances which have substantial concentrations are related to the concentration level and reaches smaller areas.

The pattern of concentration area coverage is for the pattern to increase both in area coverage and concentration levels immediately after munition function. The peak concentration level is achieved within seconds to minutes of munition function for volatile agents and within hours for intermediate volatility agents. The peak concentration levels then decrease until there is no longer any measurable levels of concentration. The area coverage for high volatility agents will tend to increase rapidly as the agent diffuses and decrease rapidly as the peak concentration decreases based on the agent diffusion. The intermediate volatility agents will tend to increase relatively slowly over a longer period of time as secondary evaporation provides new vapor to diffuse, and then slowly constrict as the agent evaporative flux decreases over time.

Concentration area coverage charts may be used to identify the peak vapor exposure that material must withstand to be survivable. In addition, the lines at different times provide insight into the concentrations that exist as well as the time frame for which they dominate. One very important function of the concentration area coverage chart can be to provide one measure of "all-clear" estimation. For most agents, the vapor hazard to eye and respiratory systems is of vital importance. If the vapor concentration drops below the level necessary to cause harm, then masks (and of course protective overgarments) can be removed. Use of

these charts to estimate the predicted time of safe unmasking can be used as a planning tool. The concentration area coverage charts can be used to project time in MOPP 4 for operational planning and focus use of detectors for "all-clear" assessments.

Use of Dosage Area Coverage Charts

The dosage area coverage charts show the vapor dosage at times intervals following an attack. Note that the graph shows the area coverage in square meters and dosage in milligrams-minutes per cubic meter. Dosage is the time-integrated concentration history. That means that not only the dosage level but the area covered at that dosage level should always be non-decreasing. Both the area coverage and the dosage level are logarithmically scaled on these charts. A series of lines relates the area covered by at least the dosage level on the horizontal axis at specific times after the attack. Dosage area coverage curves can be used to characterize the peak dosage level to be expected on the target (at low area coverages) or the maximal extent of vapor contamination (at low dosage values). At dosages to the left of the shoulder in the curve, area coverage is relatively insensitive to changes in dosage level. However, at vapor levels to the right of the shoulder in the curve, area coverage is sensitive to changes in dosage level. Vapor levels for very volatile agents such as hydrogen cyanide (AC), phosgime oxime (CX), phosgene (CG), and sarin (GB) can reach high levels of dosage and area coverage very rapidly.

Vapor levels for intermediate volatile agents such as tabun (GA), soman (GD), mustard (HD), lewisite (L), and GF tend to generate vapor dosage levels which take hours or days to reach the full potential generated by an attack.

The time necessary for total dosage levels to be achieved after an attack are a critical measure of the volatility of the agent and, by subtraction, the residual hazard which will require longer stays in full protective posture.

Use of Chemical Casualty Charts

Casualty charts are stacked bar charts which show the severity of injury potentially caused by an attack with a given level of medical therapy available. Note that the graph shows the casualty area coverage in square meters and that the axis is logarithmically scaled. Medical therapy options displayed include no medical therapy, atropine and 2-PAM, and pyridostigmine pretreatment with atropine and 2-PAM for nerve agent attacks. Medical therapy options displayed include no medical therapy and topical skin protectant for blister agent attacks. The results are presented for different protective postures. The first four bars reflect the results of no protective equipment (MOPP 0), wearing the protective overgarment (MOPP 2), wearing full protective equipment [overgarment, boots, gloves, mask, hood] (MOPP 4), and wearing the mask only. The first four bars depict maintaining these protective postures before, during, and after the attack. In other words, the MOPP 0 case

shows the consequence of using no protective equipment while the MOPP 4 case shows the consequence of using protective equipment throughout the period of hazard. The MOPP 2 case shows the incremental value of the overgarment versus no protection while the mask only case shows the value of eye-respiratory protection versus no protection. The next four bars (on all charts except VX) show the results of masking enroute to MOPP 4 for airmen at various times after munition function, i.e., the label 0=>4 @ 60 seconds is defined to mean that the airmen started in MOPP 0, the mask was put on at 60 seconds after the munition functioned, and the airmen continued to full MOPP 4 protection. It is possible to view the rise in casualty area coverage at additional periods of delay in masking as an indication of the risk inherent in delayed masking.

The lowest identified level of injury is the threshold level which reflects the beginning of the signs or symptoms of injury. For instance, in the case of nerve agents, threshold level effects include increased sweating, minor skin twitching, hoarseness, and minor eye effects. In the case of blister agents, this may include skin redness or inflammation of the eyes. This will always have the highest area coverage since these effects occur at the lowest level of injury.

The next most severe level of injury is identified as vision-impaired. This level reflects the point at which nerve agents would cause pin-point pupils, eye-lid twitching, and potentially intense pain upon exposure to sunlight. The level chosen reflects effects which would be expected to effect ground crew tasks, but that the consequences might prevent air crews from performing their mission. Potentially associated with this level of injury is the use of the personnel antidote kit containing atropine and 2-PAM. Use of atropine could cause the lenses and iris of the eye to become fixed which would interfere with reading, make focusing on objects difficult, and make the airman very sensitive to bright lights. Blister agents might cause the normally clear cornea to become cloudy and create a state of temporary blindness which might last for hours or days which would equally debilitate air and ground crews.

The incapacitation severity level represents very serious injuries. This level is set to indicate that militarily significant performance will not be possible. In the case of nerve agents, nausea, vomiting, muscular twitching, loss of bladder control, emotional disturbances, sleeplessness, convulsions, and even potential respiratory arrest depending on the severity of exposure and individual differences. A significant percentage of those people listed as incapacitated may need respiratory support to survive the exposure. Blister agents cause incapacitation by blindness, blisters in the respiratory track which cause difficulty breathing and coughing, blisters on the face which interfere with the ability to wear the protective mask, and a combination of number and location of blisters which prevent accomplishment of military jobs.

The final level of severity is the lethal level.

MORTAR

Mustard

Mortar - Mustard (HD)

A mortar attack consisting of 36 120 millimeter mortar rounds was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. Each round contained slightly more than 2 kilograms of mustard. The fireplan was constructed based on doctrine originated by the former Soviet Union. This attack is characteristic of techniques which might be used by a special operation team for self-defense and disruption of operations.

The peak liquid deposition from the attacks approached 10 grams/square meter with no liquid area coverage greater than 1 square kilometer under any of the three meteorological cases.

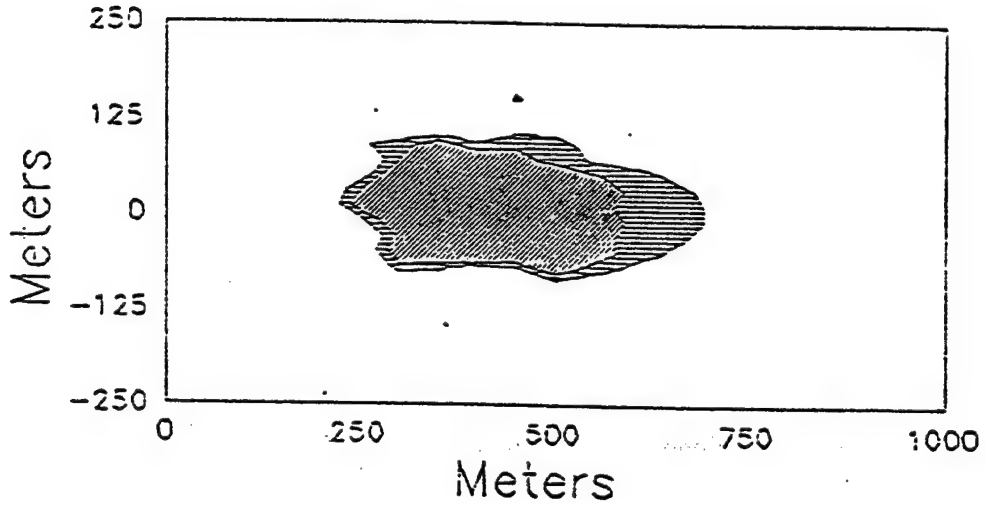
The concentration area coverage curves show that the peak concentration values were reached very early as represented by the results at 1 minute after the attack. Over time, the peak concentration diminishes while the area covered with minimal levels of concentration increases. The concentration across the target area drops below significant levels between 16 hours and 1 day for the low temperature, low windspeed case; between 1 hour and 16 hours for the moderate temperature, moderate windspeed case; and between 3 minutes and 1 hour for the high temperature, high windspeed case.

The dosage area coverage curves show very important characteristics of this attack with mustard. The Pasquill stability category E shows the highest observed dosages reaching levels approaching, but not achieving the level of dosage responsible for 50% lethality (50% lethality requires 1,500 milligram-minutes/cubic meter). Also significant is that this case results in agent dosage being carried to almost 10 square kilometers. The neutral condition of Pasquill stability category D is only able to achieve a peak dosage of slightly higher than 100 milligram-minutes/cubic meter and a maximum area coverage of less than 10 square kilometers. The Pasquill stability category B shows even lower peak dosages and area coverages (significantly lower than 100 milligram-minutes/cubic meter and less than 1 square kilometer respectively).

The casualty area coverage charts across the meteorological conditions show a relative change in the intensity of casualties which follows the drop in peak dosage area coverage. For the relatively low dosage values achieved by the level of mortar rounds used in this attack, only the low temperature, low windspeed, Pasquill stability category E resulted in potential areas of lethal effects. Even then, the level of lethal effects is only 1,000 square meters when protective mask is not worn or put on between 1 hour and 16 hours after the attack.

The moderate temperature, moderate windspeed, Pasquill stability category D results in almost no levels of incapacitation unless masking is delayed beyond several minutes. The high temperature, high windspeed, Pasquill stability category B resulted in only threshold level effects for all practical purposes.

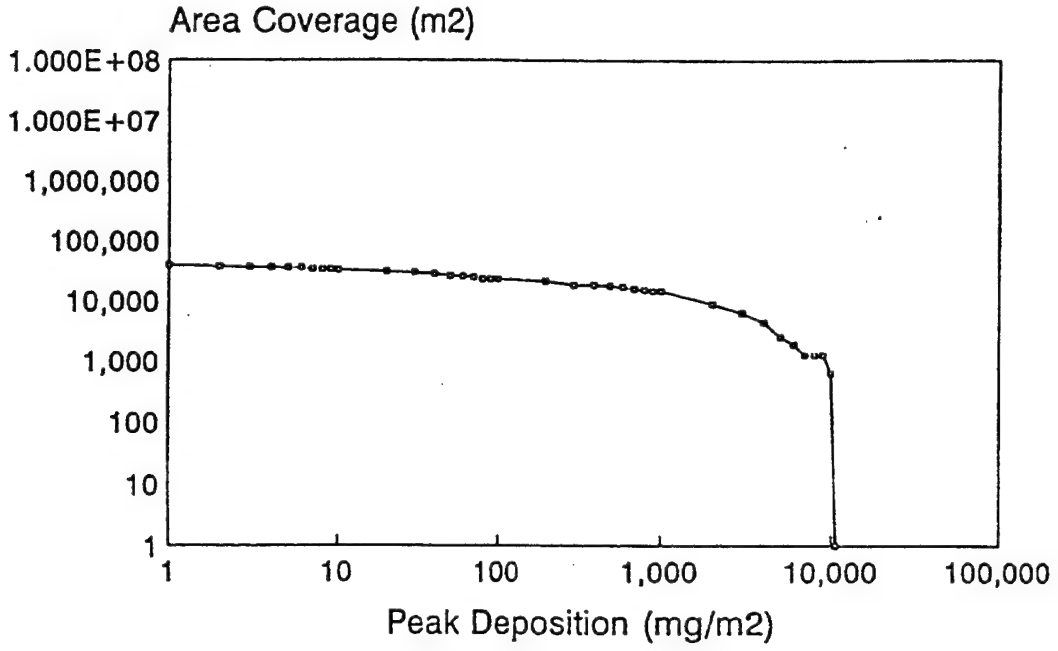
Thirty-six 120mm Rounds
Mustard (HD)



4oC (40oF)
1.5 m/sec
Stability E

Visually Impaired
Incapacitated
Lethal

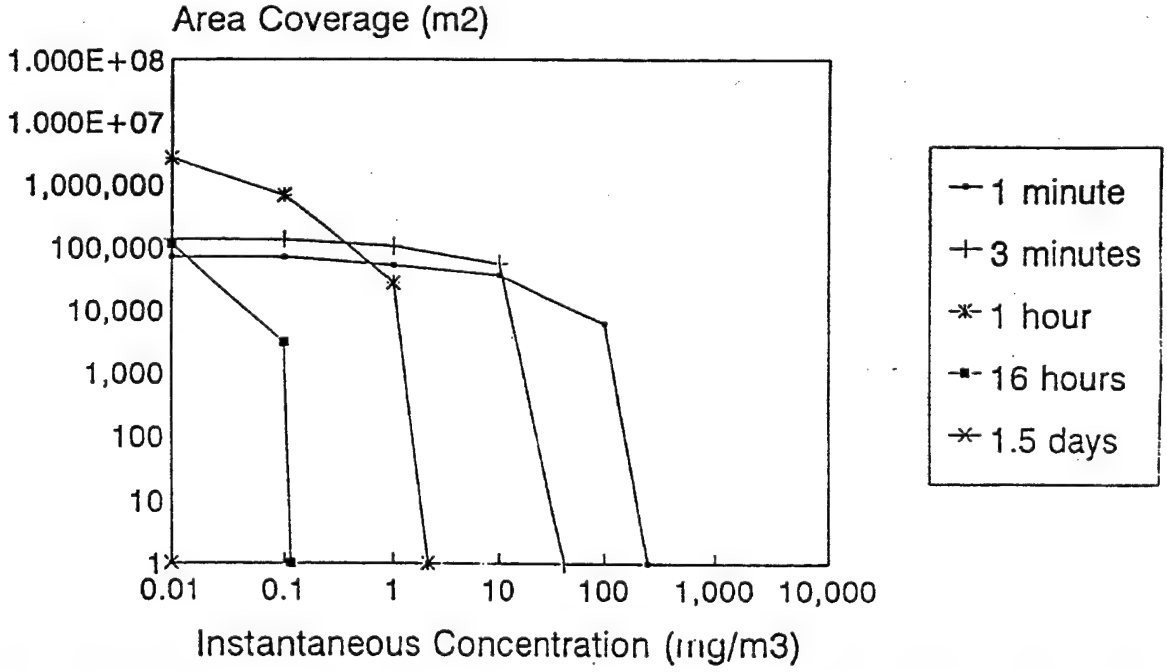
Thirty-Six 120-mm Mortar Rounds Mustard (HD)



3-30

4°C (40°F), 1.5m/sec, stability E

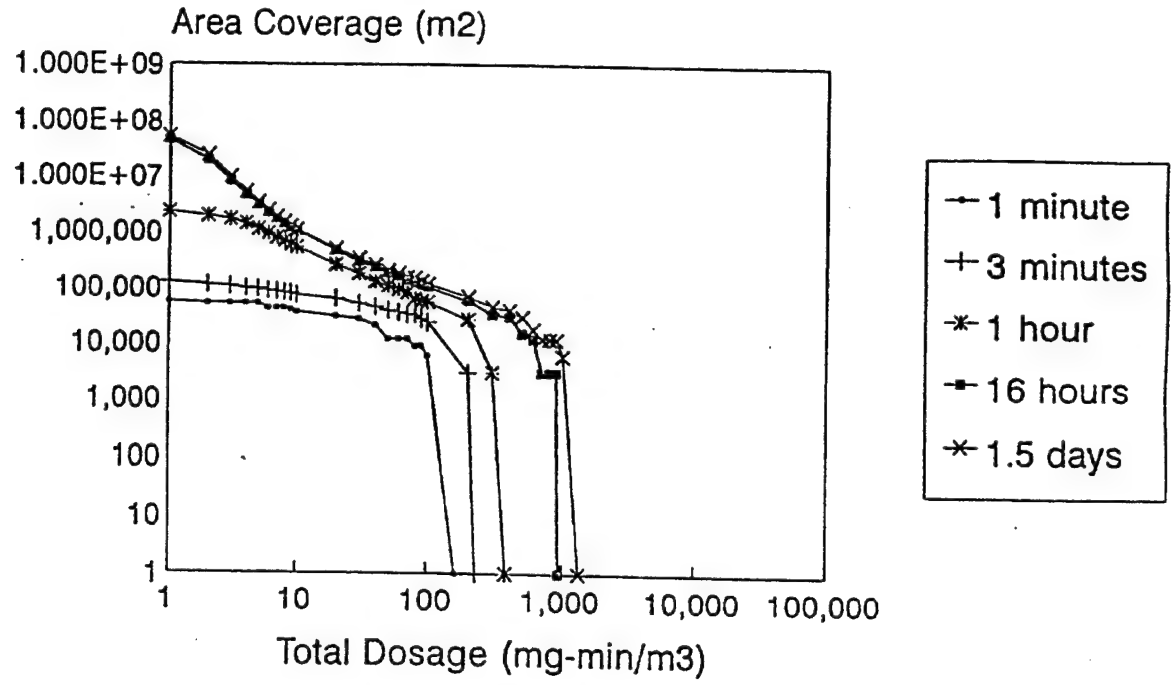
Thirty-Six 120-mm Mortar Rounds Mustard (HD)



3-31

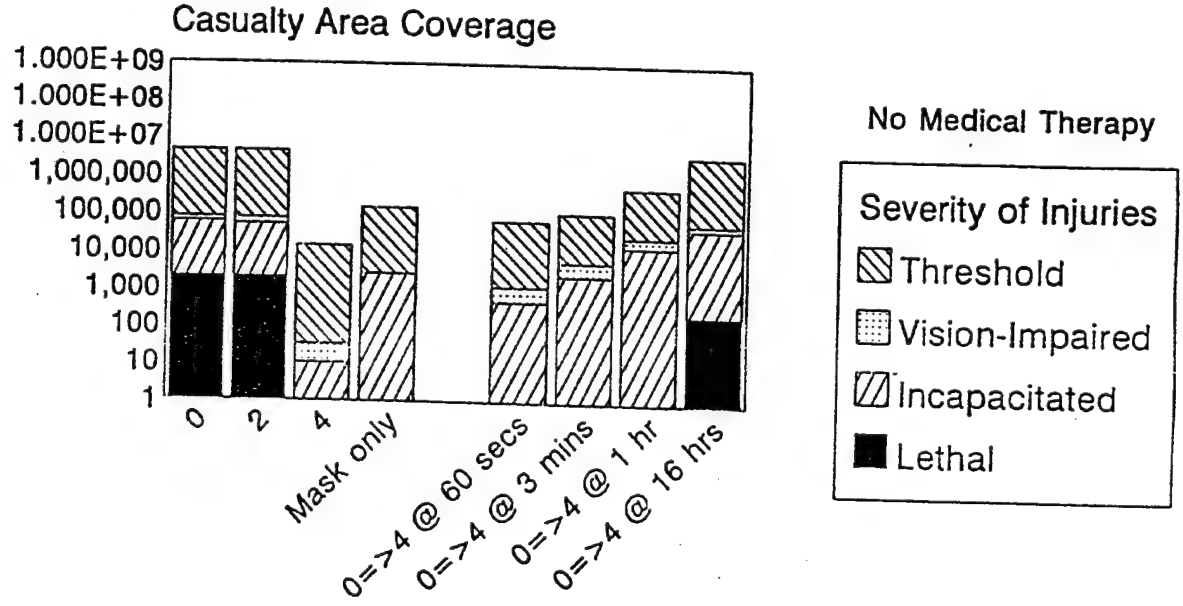
4°C (40°F), 1.5m/sec, stability F

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



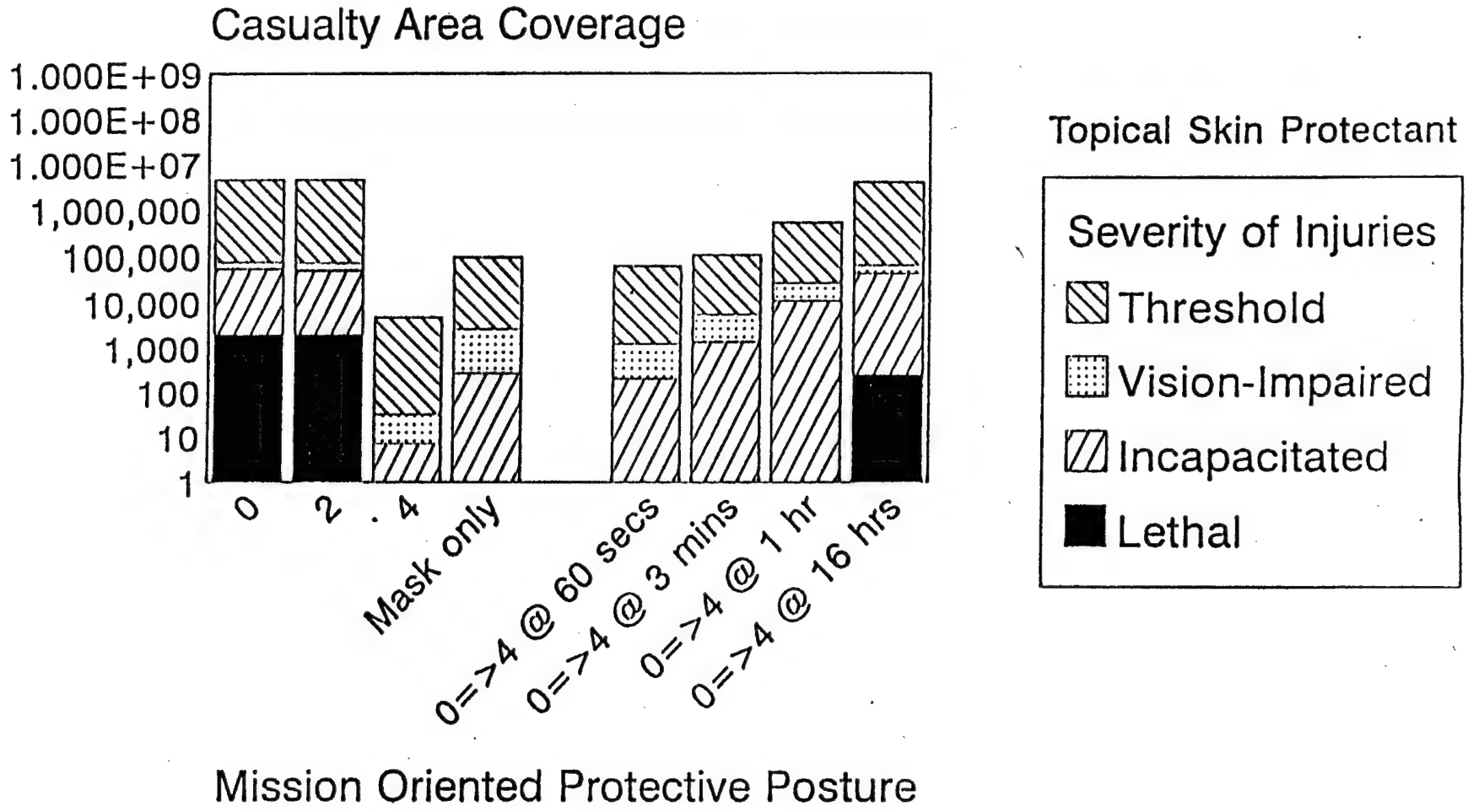
4°C (40°F), 1.5m/sec, stability E

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



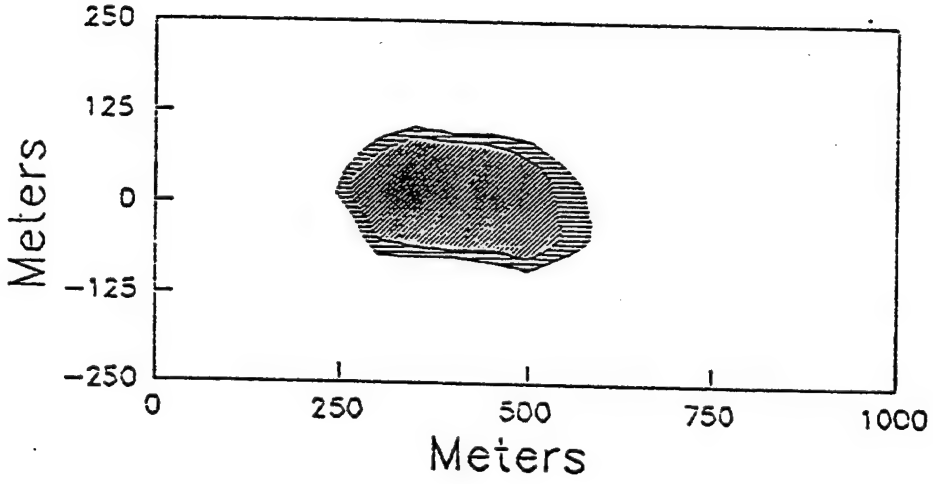
4°C (40°F), 1.5m/sec, Stability E

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



4°C (40°F), 1.5m/sec, Stability E

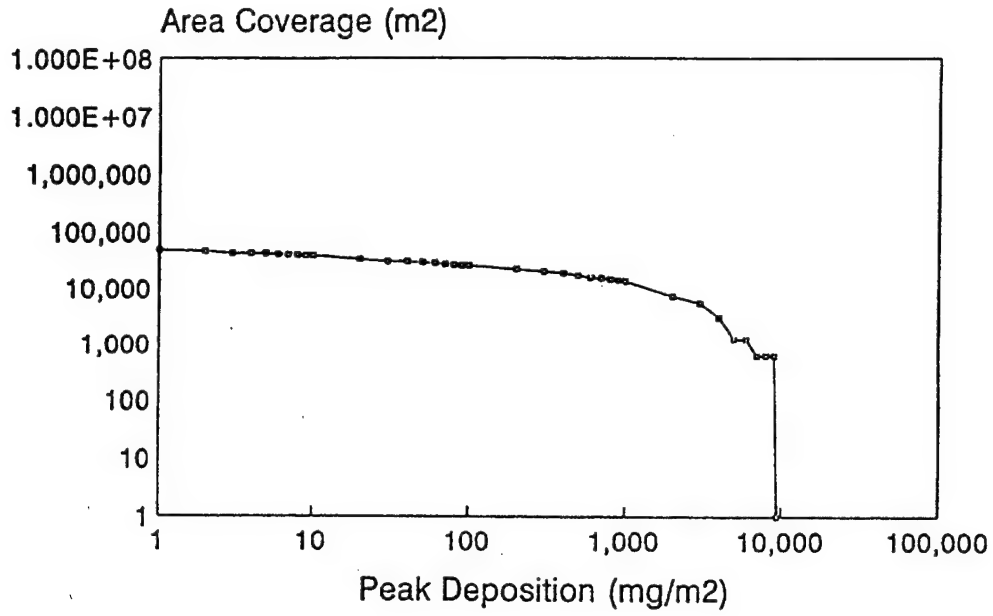
Thirty-six 120mm Rounds
Mustard (HD)



25oC (77oF)
3 m/sec
Stability D

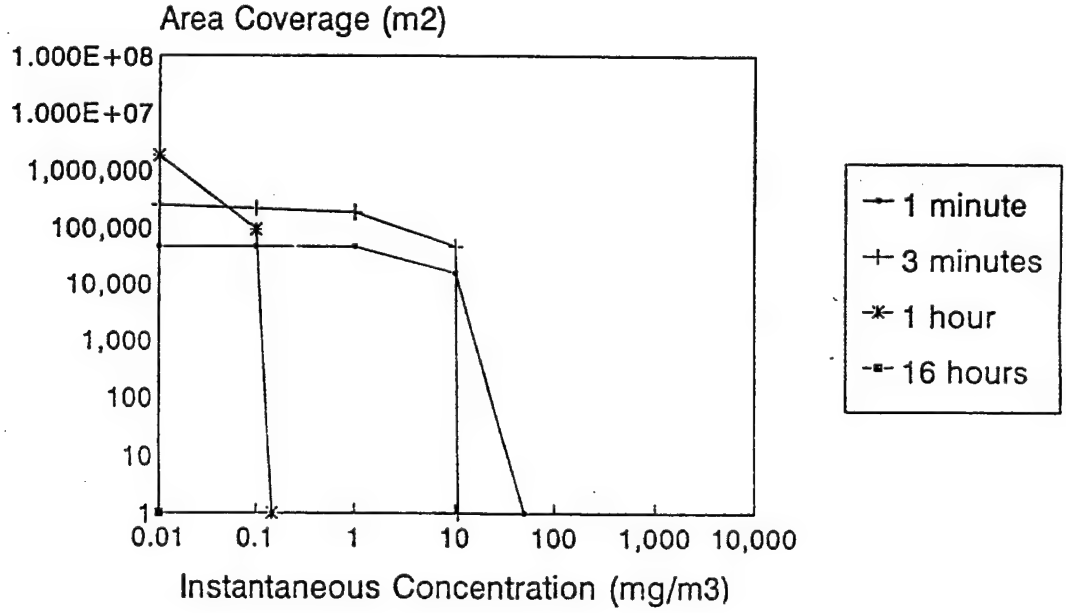
Visually Impaired
Incapacitated
Lethal

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



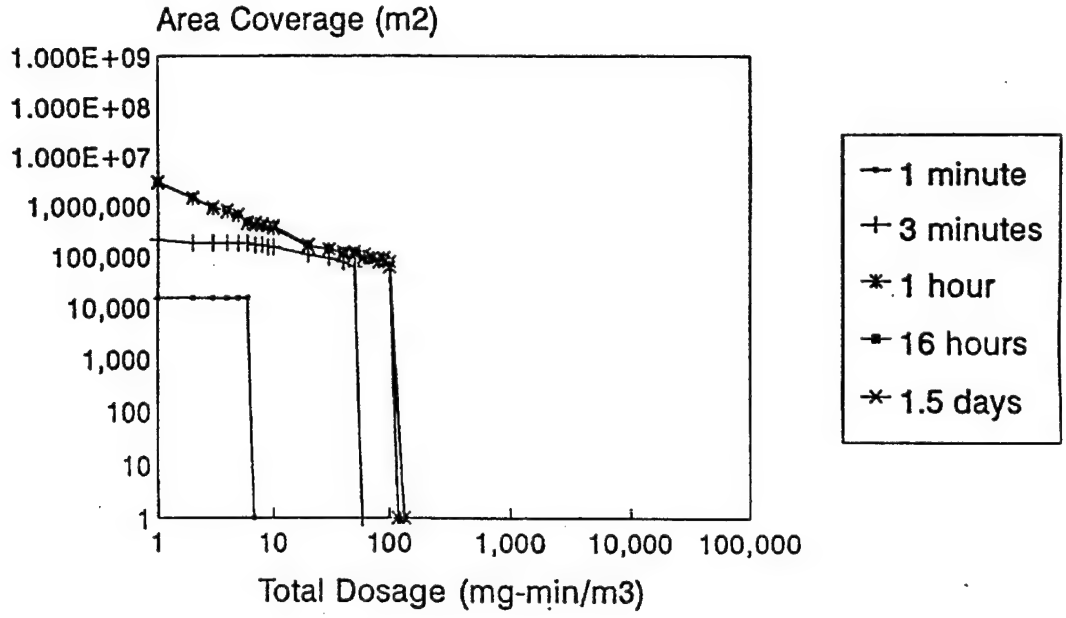
25°C (77°F), 3m/sec, stability D

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



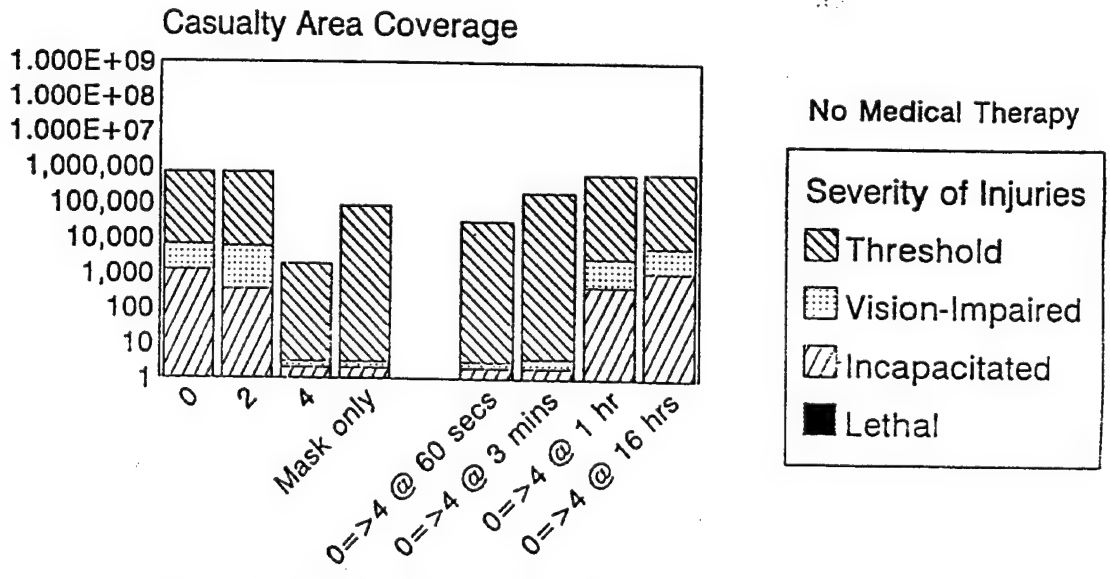
25°C (77°F), 3m/sec, stability D

Thirty-Six 120-mm Mortar Rounds
Mustard (HD)



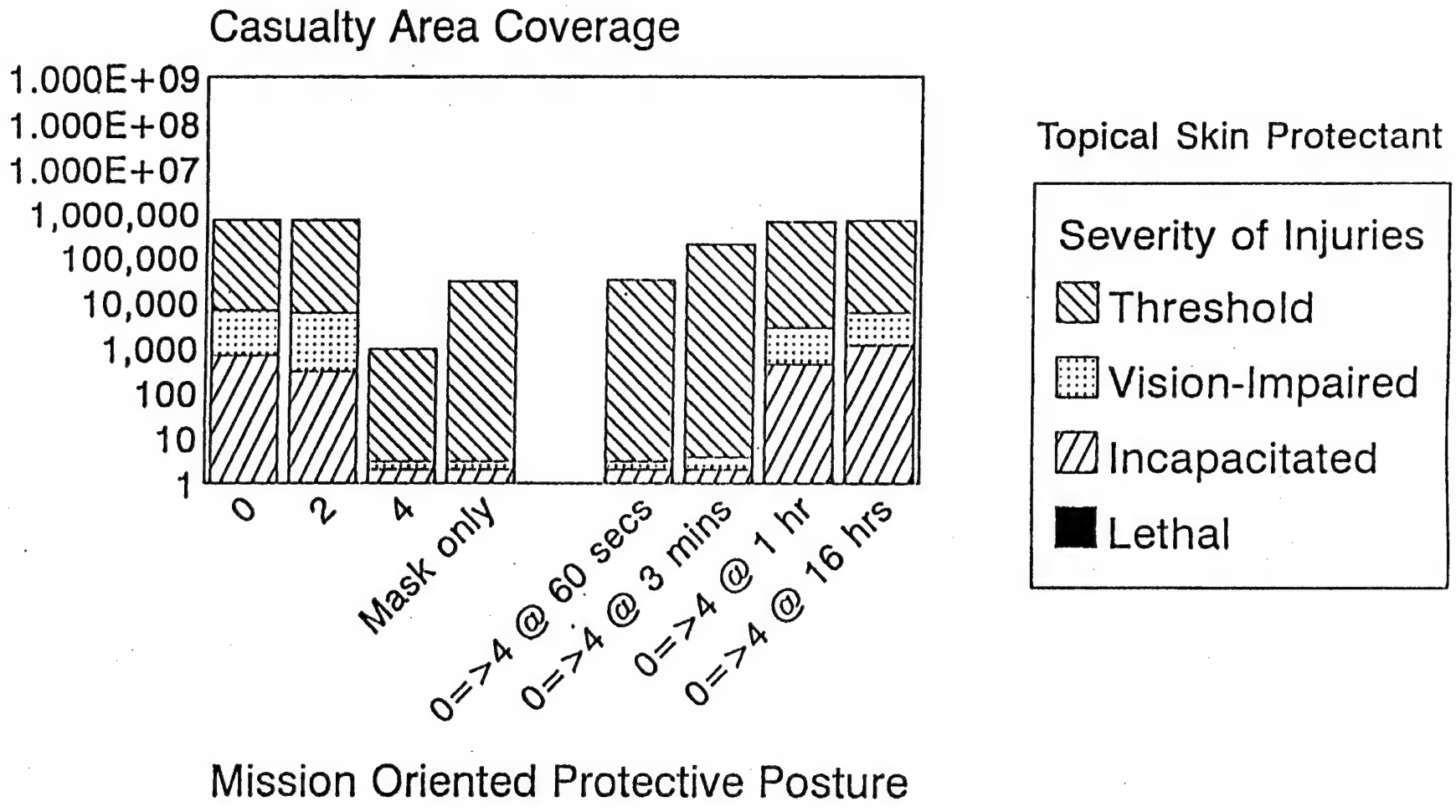
25°C (77°F), 3m/sec, stability D

Thirty-Six 120-mm Mortar Rounds
Mustard (HD)



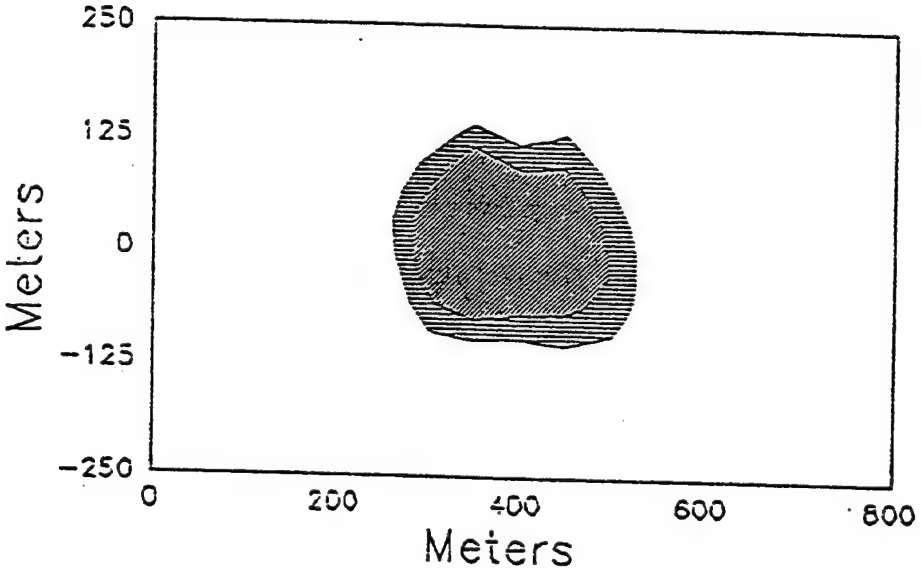
25°C (77°F), 3m/sec, Stability D

Thirty-Six 120-mm Mortar Rounds
Mustard (HD)



5°C (77°F), 3m/sec; Stability D

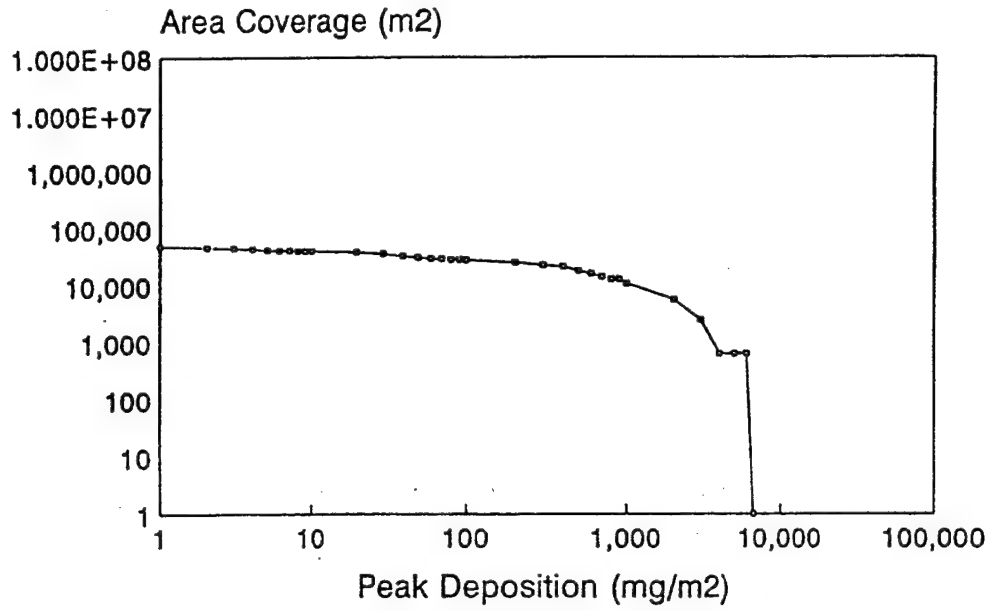
Thirty-six 120mm Rounds
Mustard (HD)



49oC (120oF)
6 m/sec
Stability B

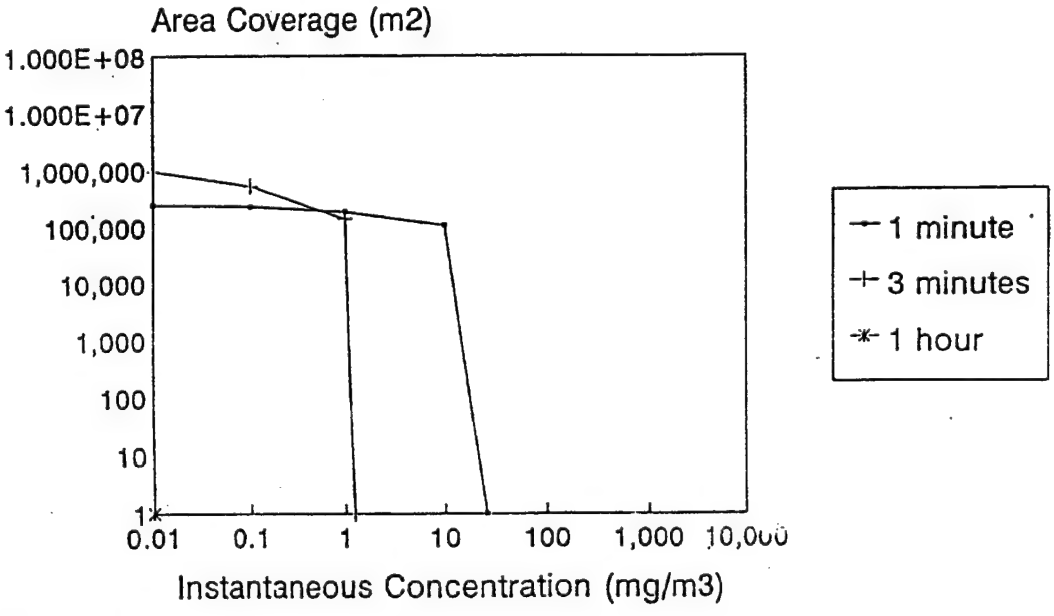
Visually Impaired
Incapacitated
Lethal

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



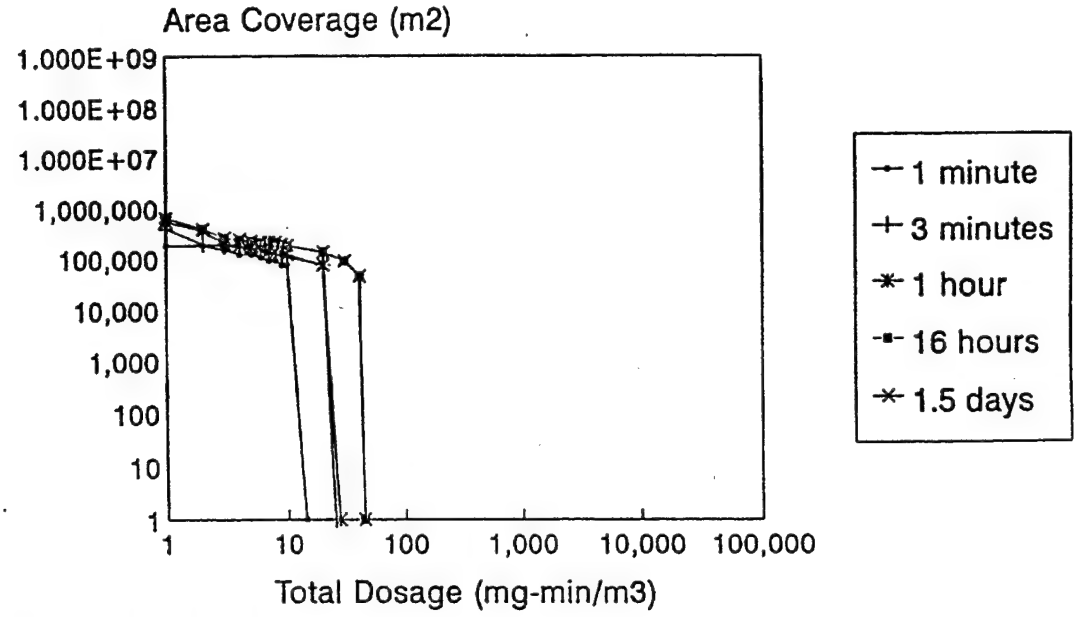
49°C (120°F), 6m/sec, stability B

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



49°C (120°F), 6m/sec, stability B

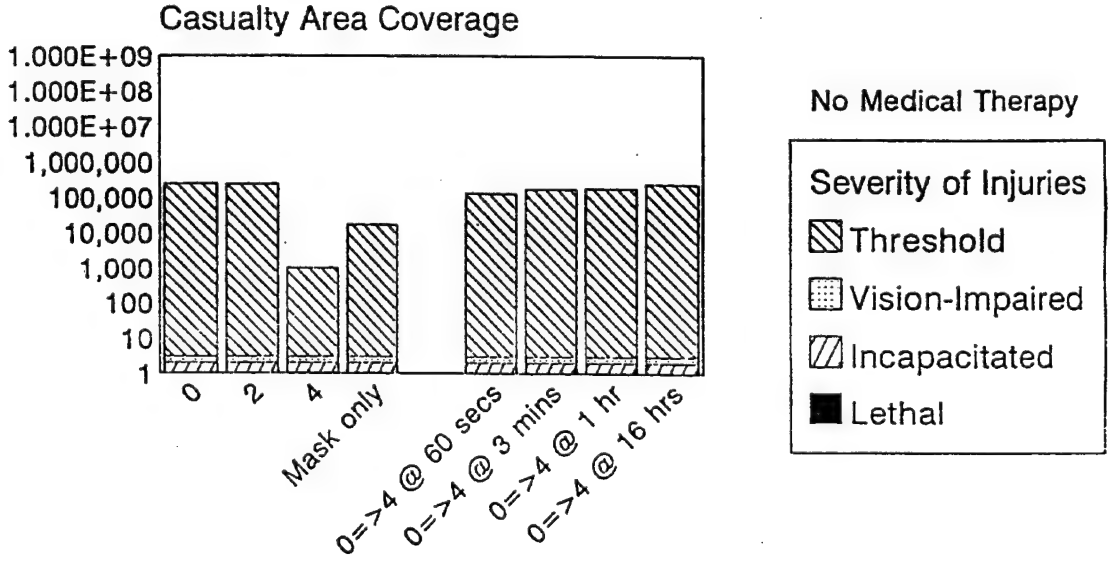
Thirty-Six 120-mm Mortar Rounds Mustard (HD)



3-44

49°C (120°F), 6m/sec, stability B

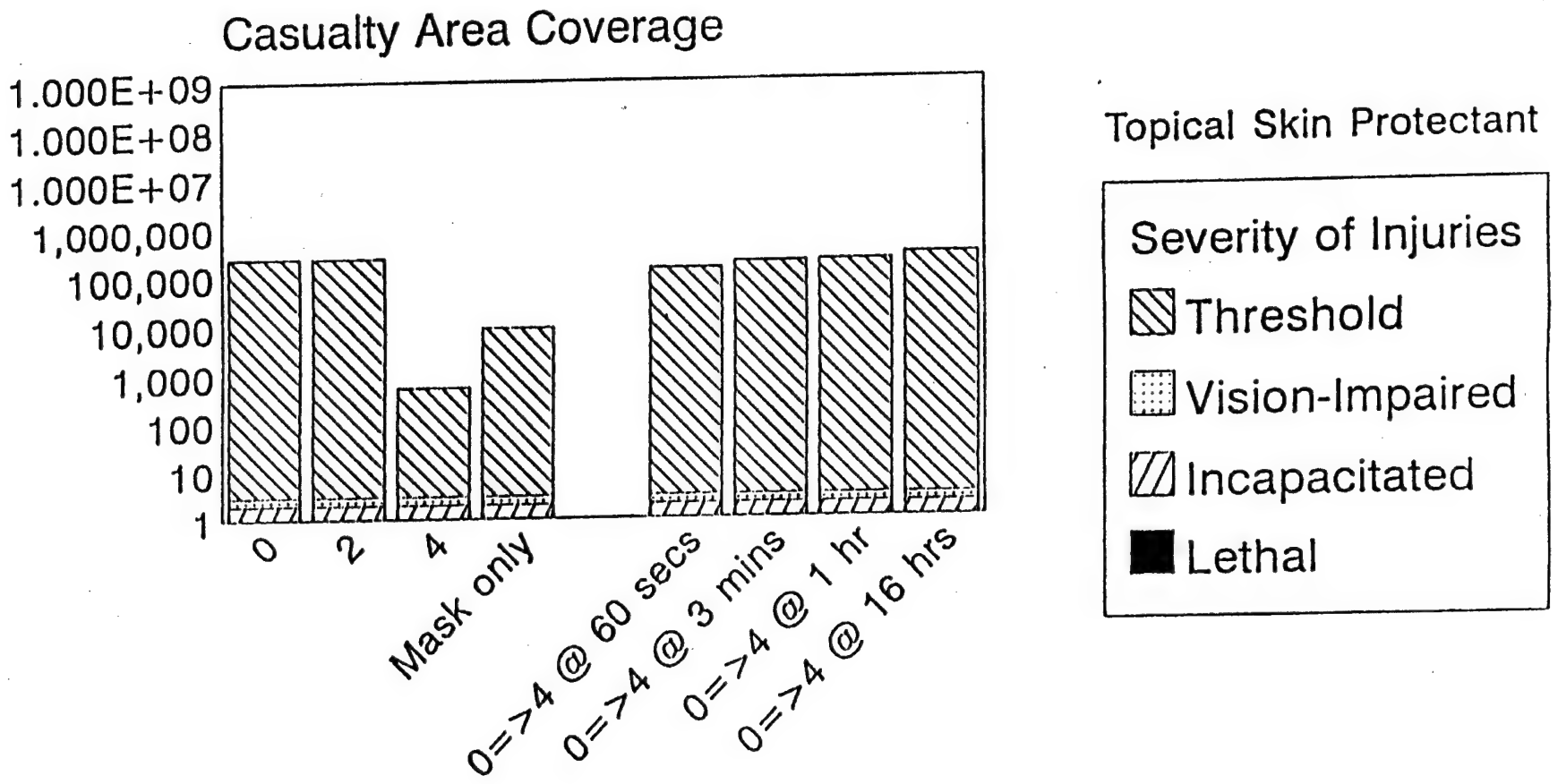
Thirty-Six 120-mm Mortar Rounds Mustard (HD)



3-45

Mission Oriented Protective Posture

Thirty-Six 120-mm Mortar Rounds Mustard (HD)



49°C (120°F), 6m/sec, Stability B

MODERATE RANGE SYSTEMS

Bomb - Sarin (GB)

Bomb - Mustard (HD)

Bomb - Thickened Soman (TGD)

Tactical Ballistic Missile with Submunitions - Sarin (GB)

Tactical Ballistic Missile with Submunitions - Soman (GD)

Tactical Ballistic Missile with Submunitions - VX

Tactical Ballistic Missile - Sarin (GB)

Tactical Ballistic - Thickened Soman (TGD)

Tactical Ballistic - Thickened VX (TVX)

BOMB

Sarin (GB)

Bomb - Sarin (GB)

An aircraft bombing attack consisting of 32 250 kilogram ground burst bombs was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. Each bomb contained slightly less than 50 kilograms of sarin. The fireplan was based on a stick release of 8 bombs on each of 4 aircraft flying nearly the same approach over the target. This attack could also be used to represent an attack by 1 bomber aircraft. Sarin is the most volatile of the nerve agents. It is not effective in long term contamination which would require prolonged operation in protective equipment; however, it does challenge the speed with which the mask must be donned.

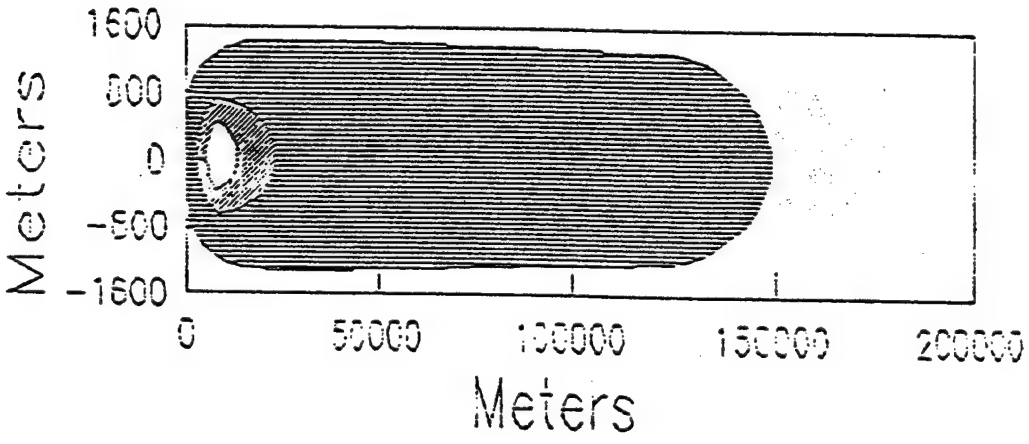
The peak liquid deposition from the attacks was considerably higher than 10 grams/square meter with no liquid area coverage greater than 1 square kilometer under any of the three meteorological cases.

The concentration area coverage curves show that the peak concentration values were reached very early as represented by the results at 30 seconds after the attack. Over time, the peak concentration diminishes while the area covered with minimal levels of concentration increases. The concentration across the target area drops below significant levels after 2 hours for the low temperature, low windspeed case and between 20 minutes and 2 hour for both the moderate temperature, moderate windspeed case and the high temperature, high windspeed case.

The inversion condition of Pasquill stability category E shows the highest observed dosages reaching levels approaching 10,000 milligram-minutes/cubic meters. Also significant is that this case results in agent dosage being carried to almost 100 square kilometers. While the neutral condition of Pasquill stability category D is only able to achieve a peak dosage of just slightly lower than the inversion condition, the maximum area coverage of less is nearly identical at almost 100 square kilometers. The Pasquill atmospheric stability category B shows even lower peak dosages and area coverages.

The casualty area coverage charts show a drop of more than an order of magnitude in lethal level effects as the temperature go from cold to hot. There is a two to three order of magnitude reduction in lethal area for wearing the protective mask. There is only a small improvement in lethal area afforded by adding the ensemble (MOPP 2 vs MOPP 0 or MOPP4 vs Mask only). The lethal area coverage shows that donning the mask at 30 seconds after the attack results in nearly a 2 order of magnitude more area coverage than occurs when the mask is on at the time of attack. Medical intervention reduces the likely lethal area by almost an order of magnitude (more than a 90% reduction).

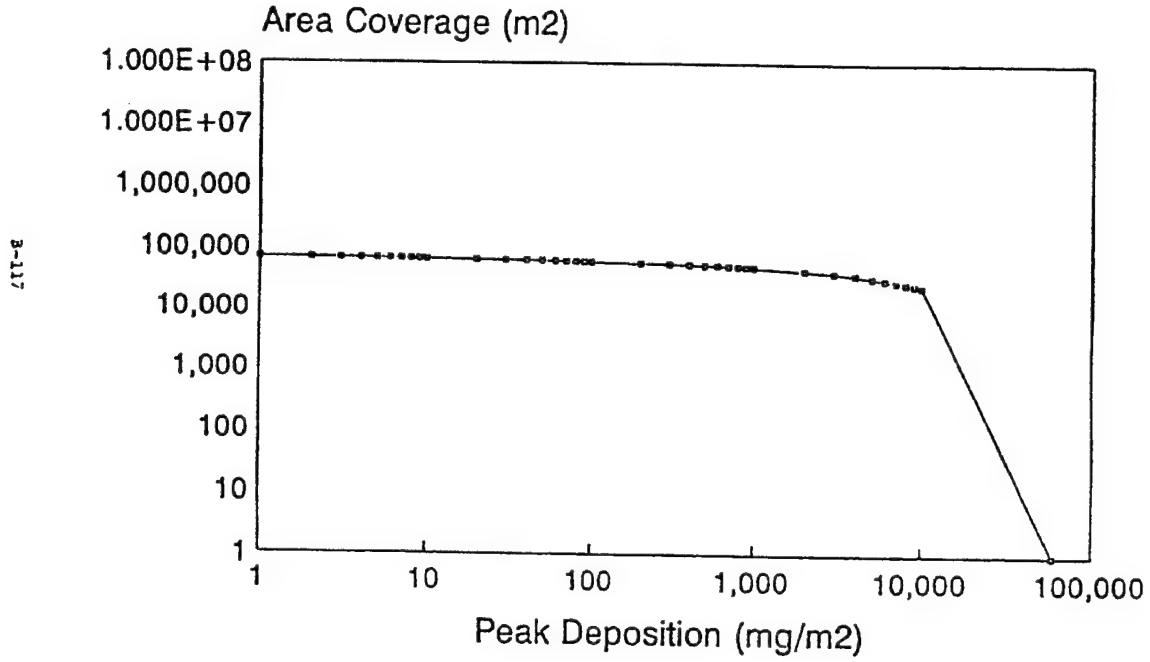
Thirty-Two 250-kg Bombs
Sarin (GB)



4oC (40oF)
1.5 m/sec
Stability E

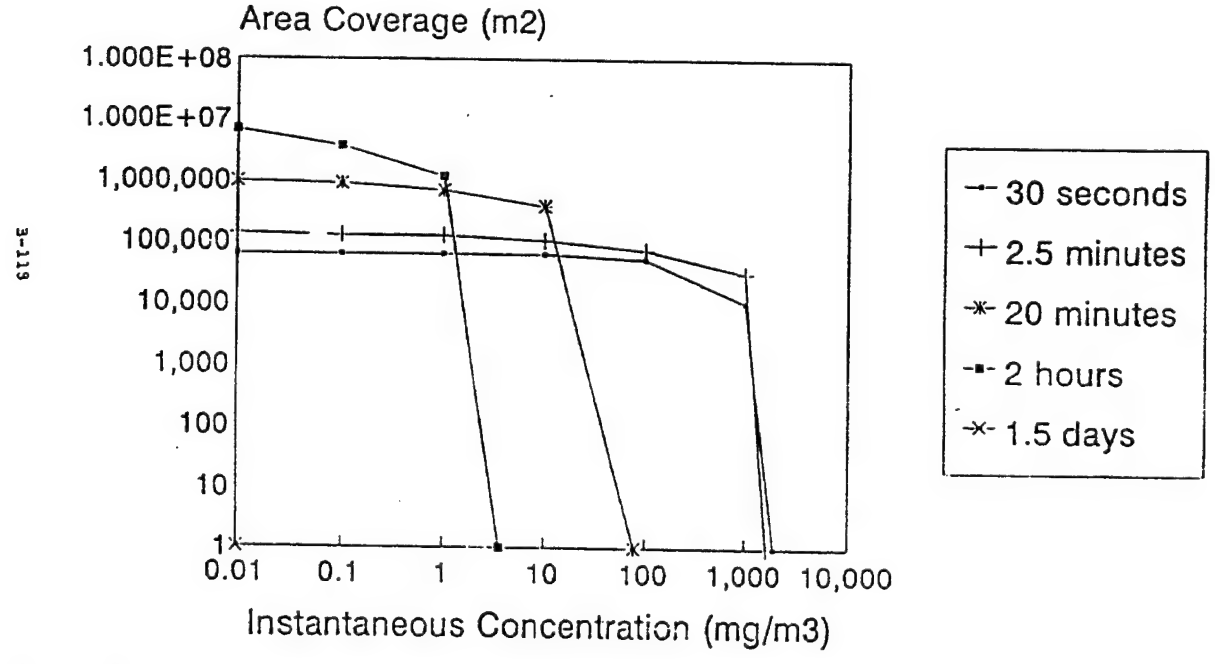
Visually Impaired
Incapacitated
Lethal

Thirty-Two 250-kg Bombs
Sarin (GB)



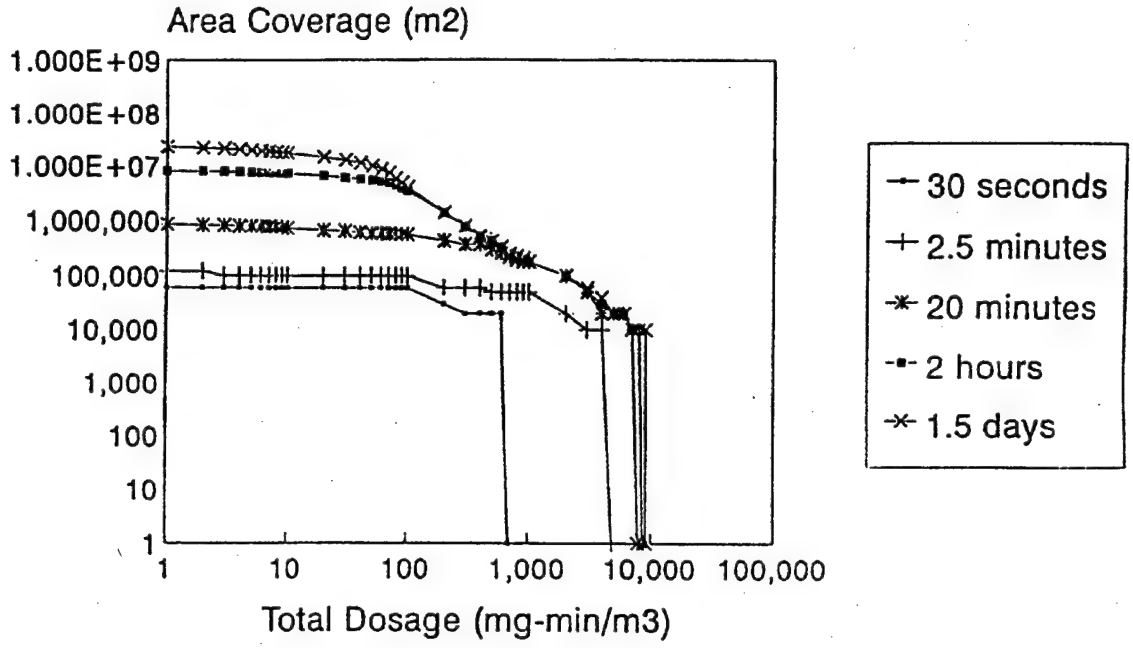
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Bombs
Sarin (GB)



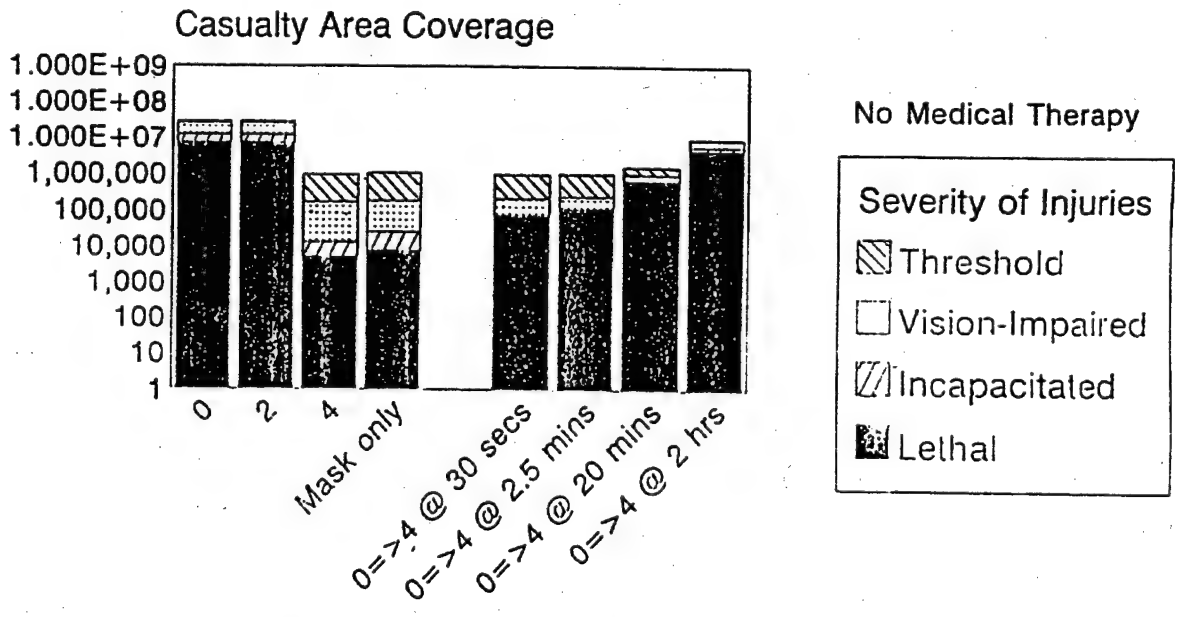
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Bombs
Sarin (GB)



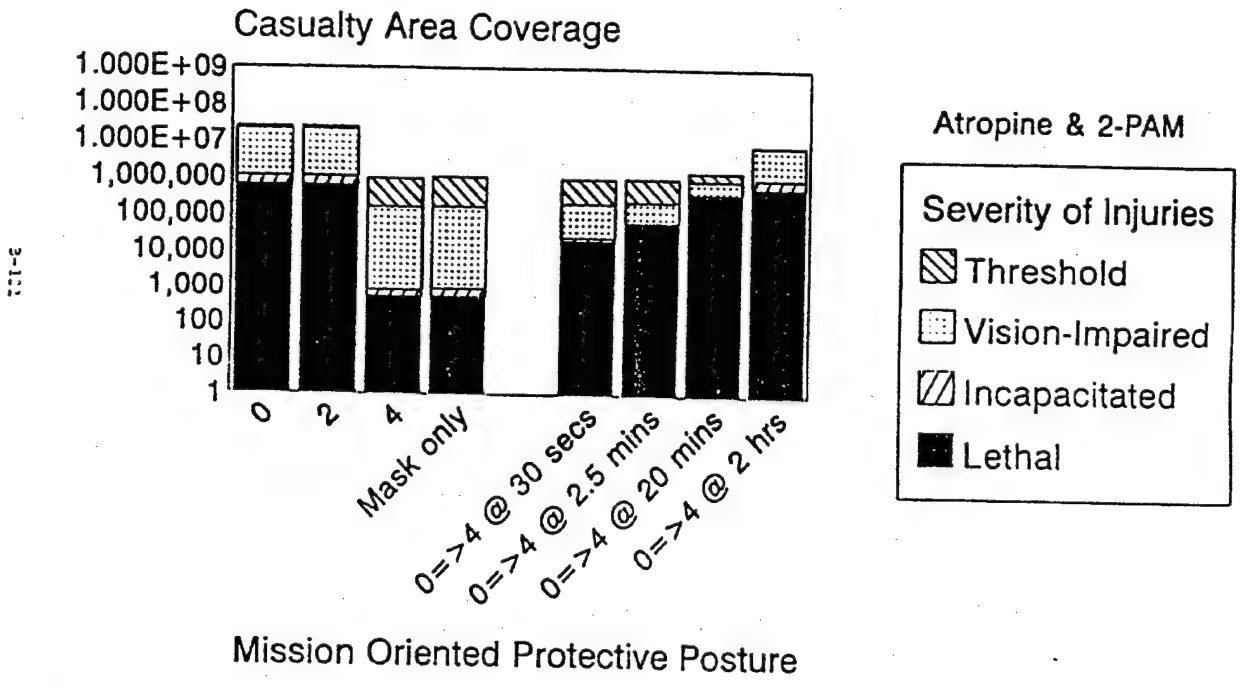
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Bombs
Sarin (GB)



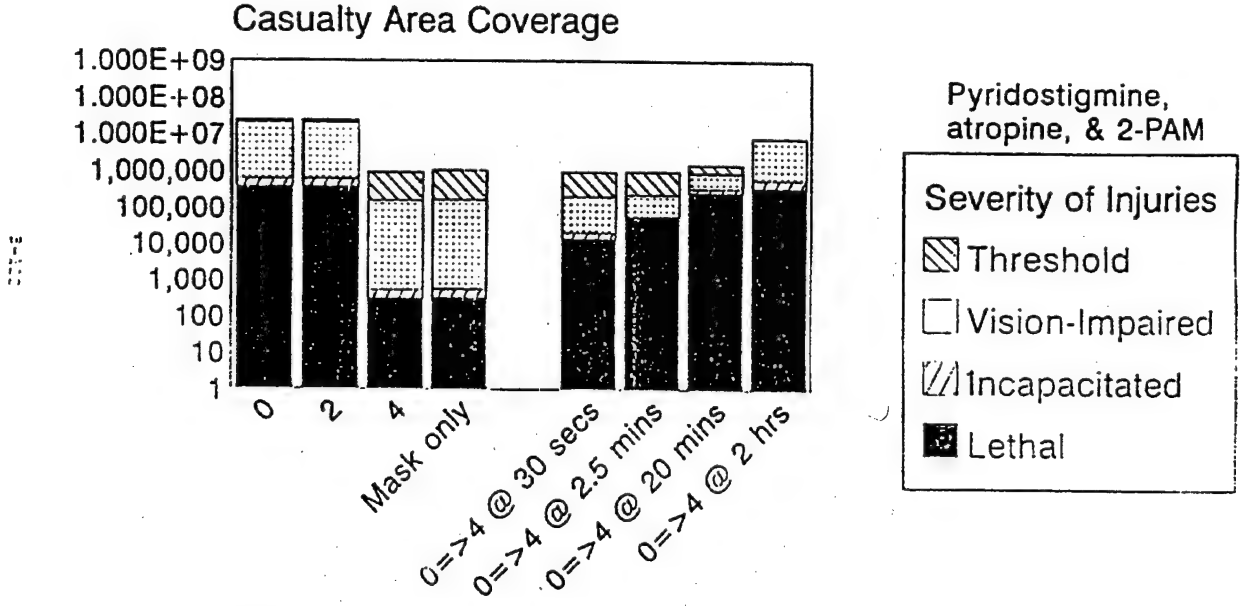
4°C (40°F), 1.5m/sec, Stability E

Thirty-Two 250-kg Bombs
Sarin (GB)



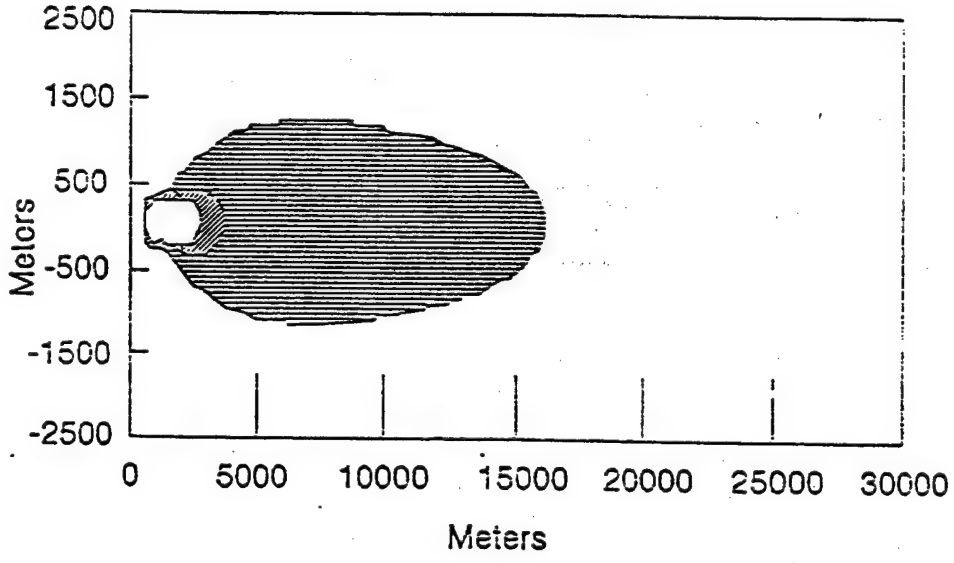
4°C (40°F), 1.5m/sec, Stability E

Thirty-Two 250-kg Bombs
Sarin (GB)



4°C (40°F), 1.5m/sec, Stability E

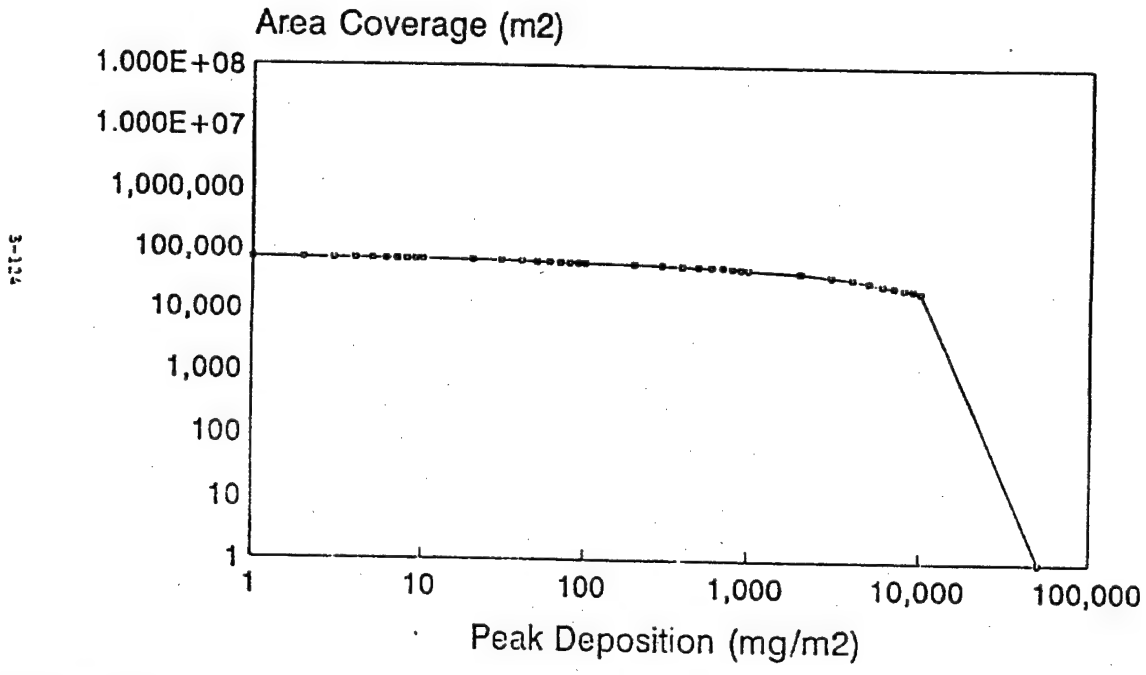
Thirty-Two 250-kg Bombs
Sarin (GB)



25oC (77oF)
3 m/sec
Stability D

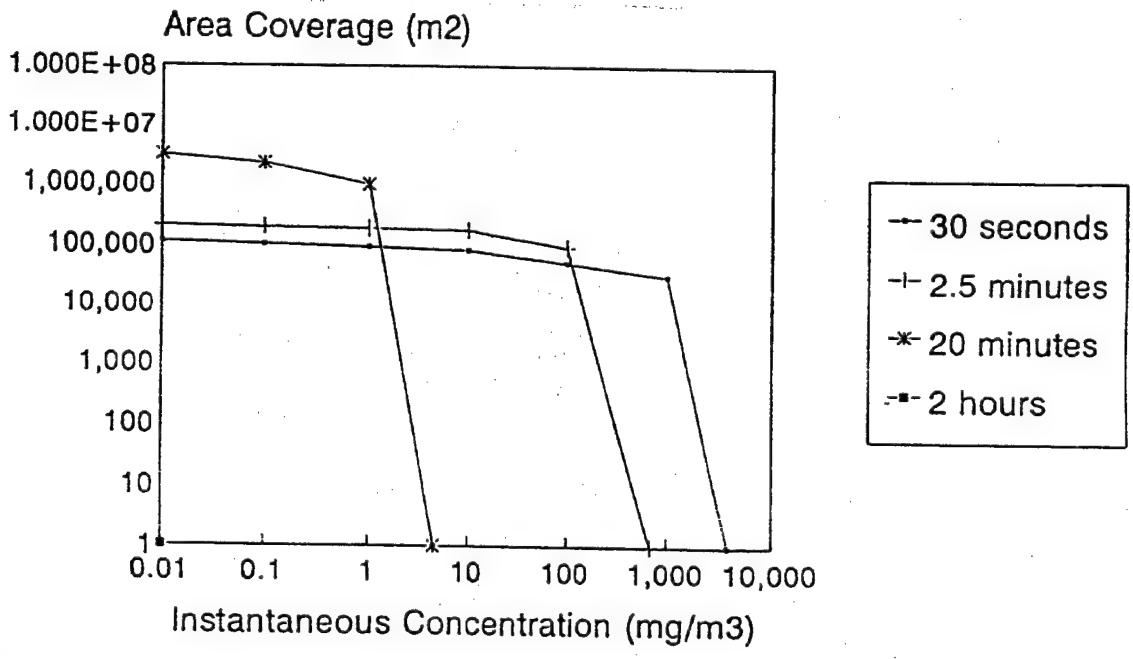
Visually Impaired
Incapacitated
Lethal

Thirty-Two 250-kg Bombs
Sarin (GB)



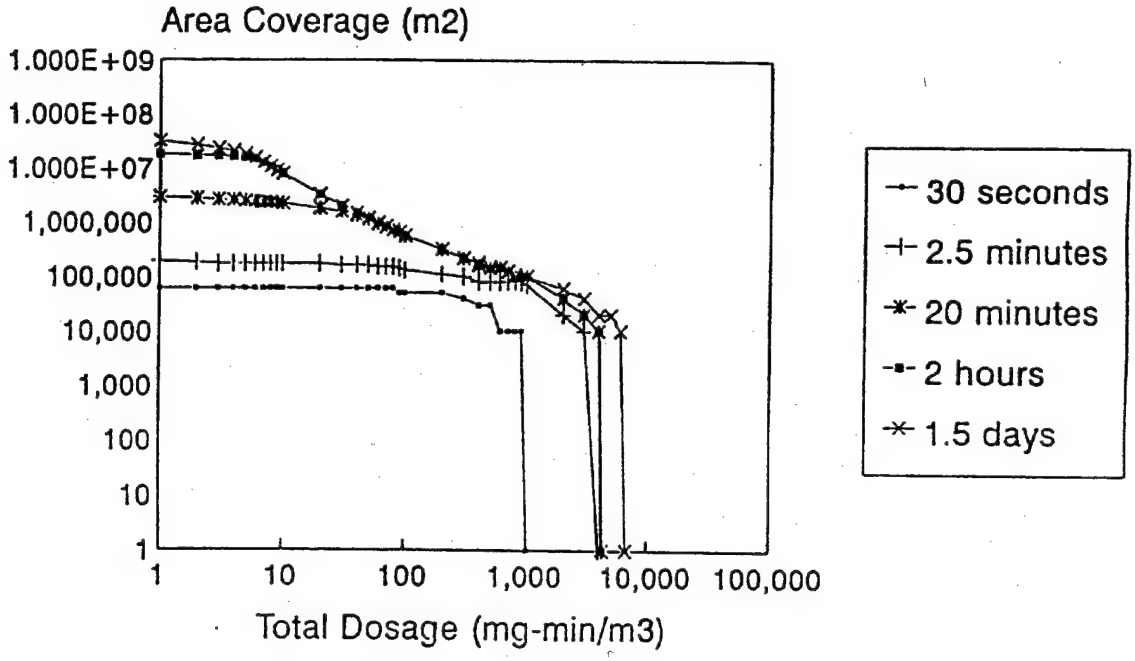
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Bombs
Sarin (GB)



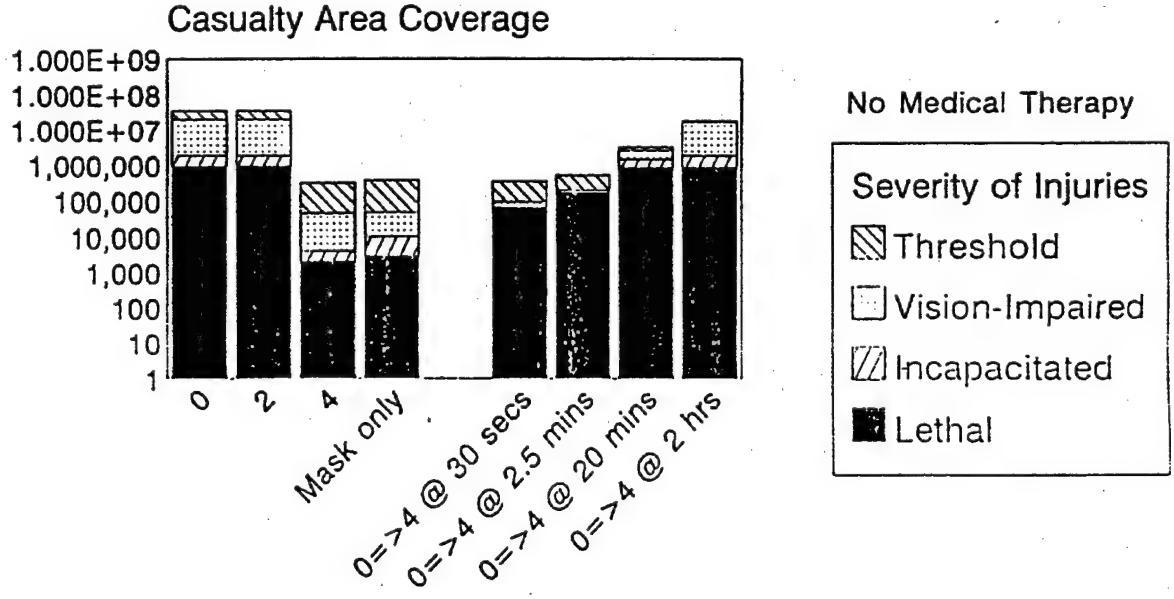
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Bombs
Sarin (GB)



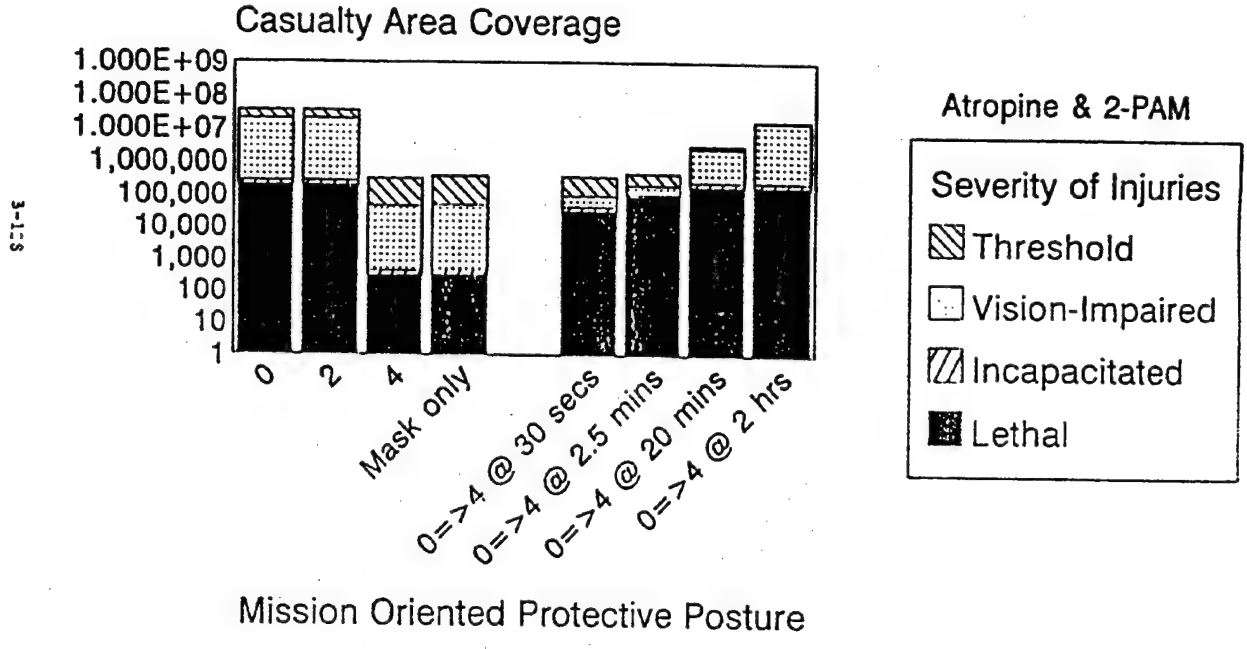
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Bombs
Sarin (GB)



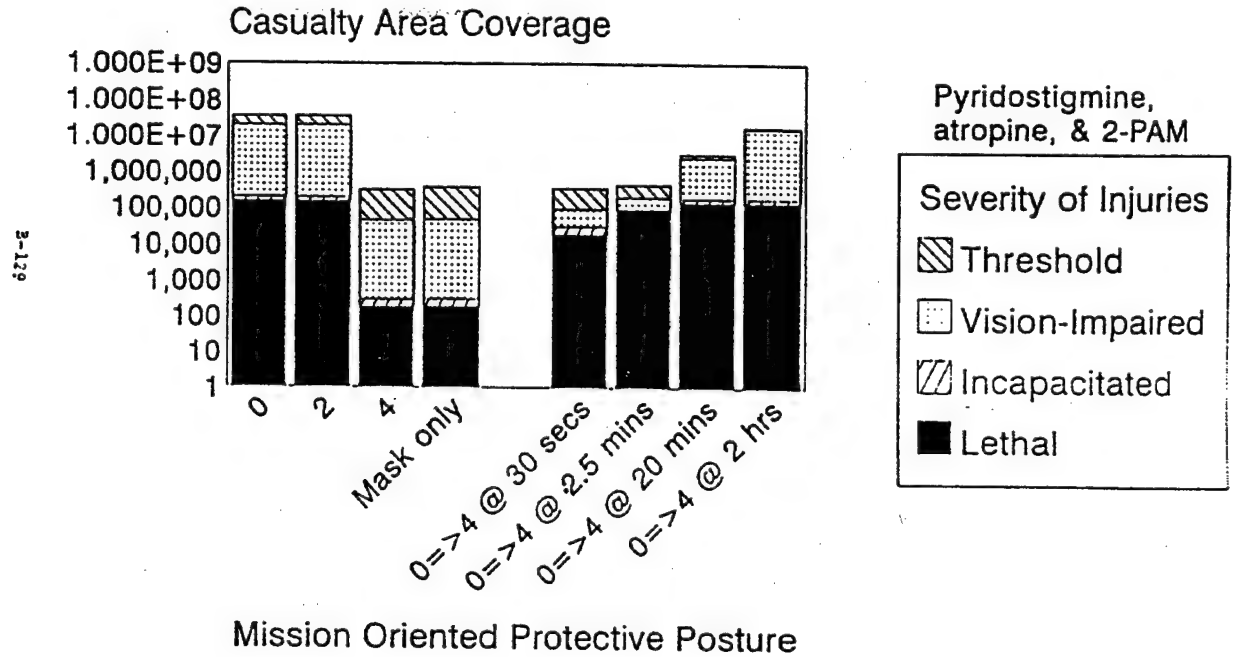
25°C (77°F), 3m/sec, Stability D

Thirty-Two 250-kg Bombs
Sarin (GB)



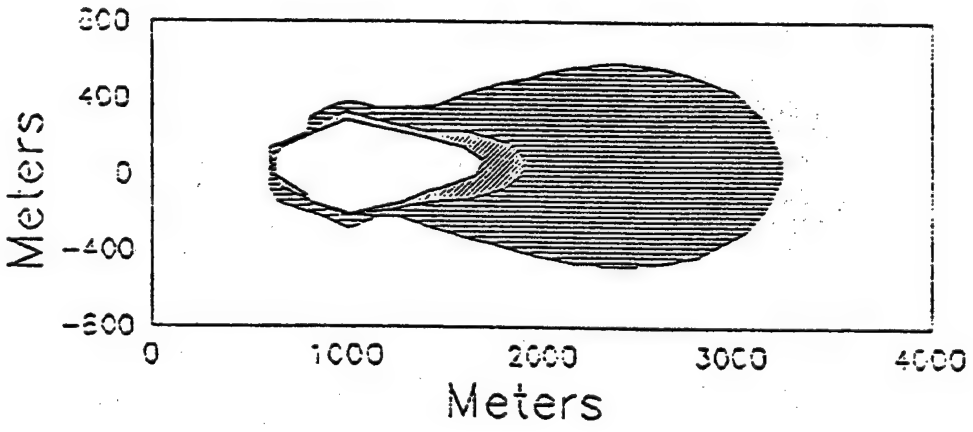
25°C (77°F), 3m/sec, Stability D

Thirty-Two 250-kg Bombs
Sarin (GB)



25°C (77°F), 3m/sec, Stability D

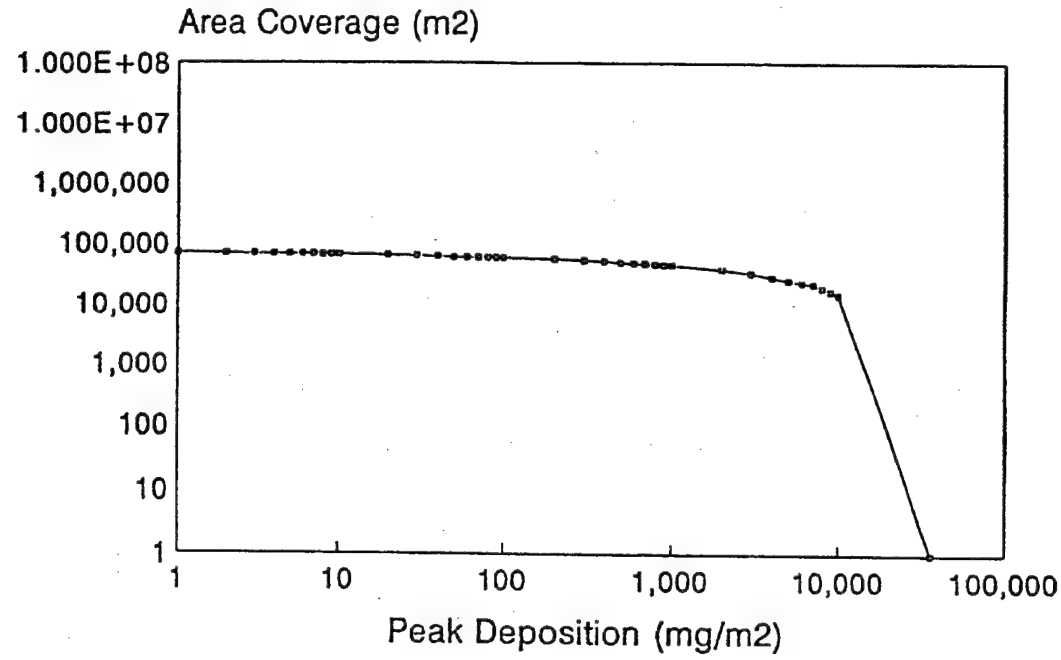
Thirty-Two 250-kg Bombs
Sarin (GB)



49oC (120oF)
6 m/sec
Stability B

Visually Impaired
Incapacitated
Lethal

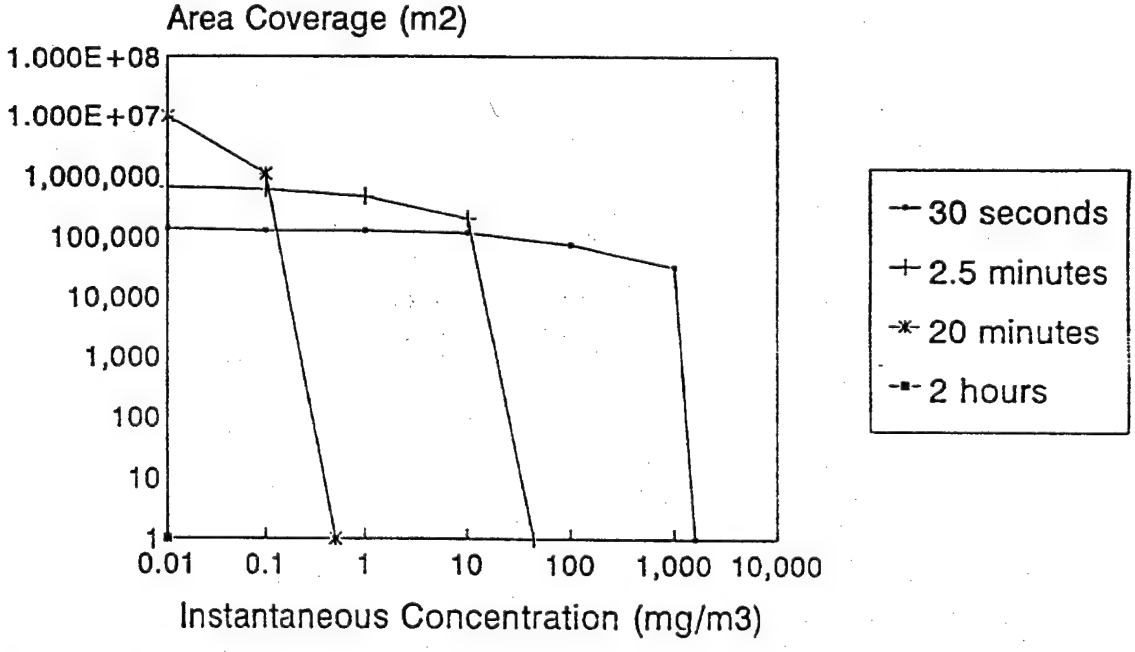
Thirty-Two 250-kg Bombs
Sarin (GB)



3-131

49°C (120°F), 6m/sec, stability B

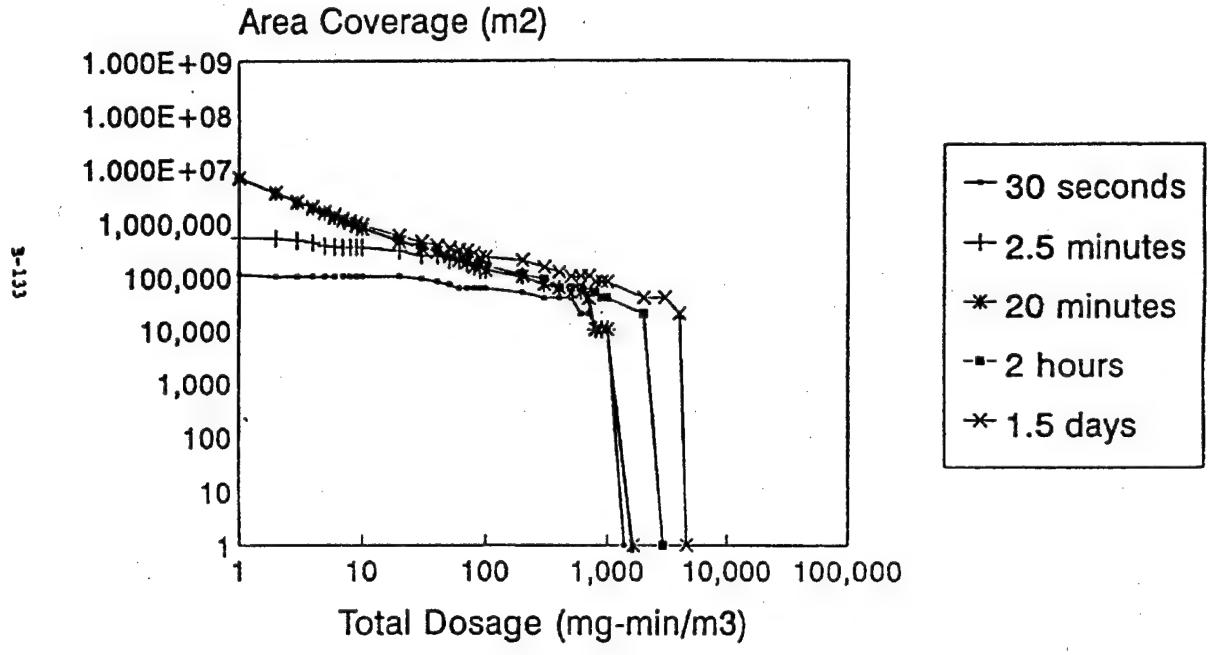
Thirty-Two 250-kg Bombs
Sarin (GB)



3-132

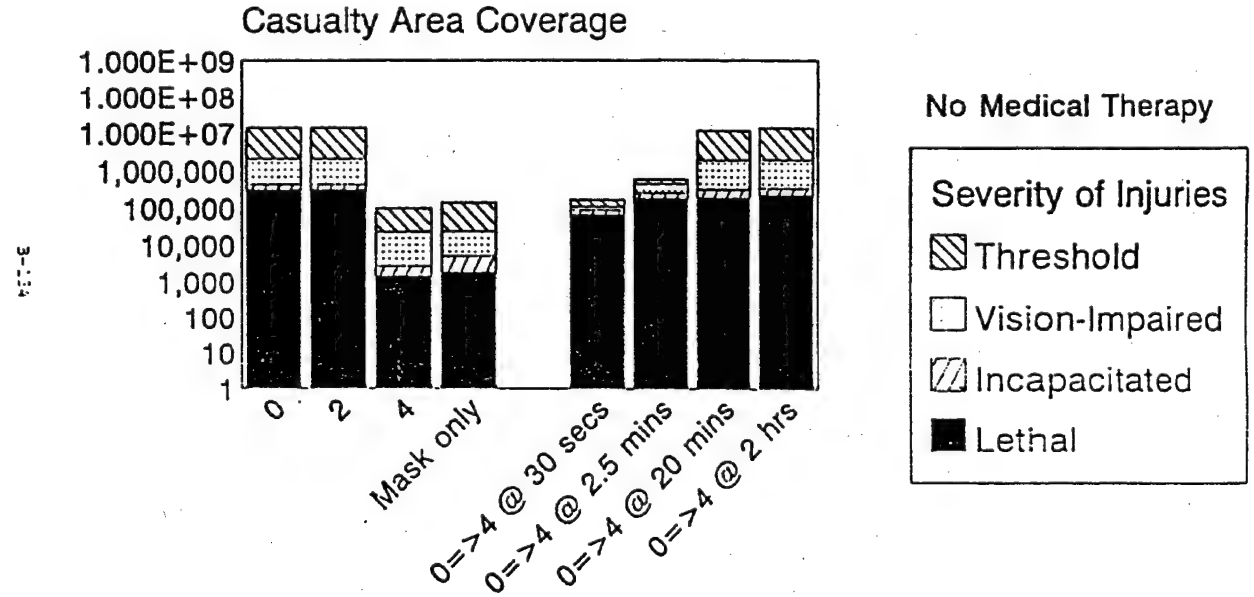
49°C (120°F), 6m/sec, stability B

Thirty-Two 250-kg Bombs
Sarin (GB)



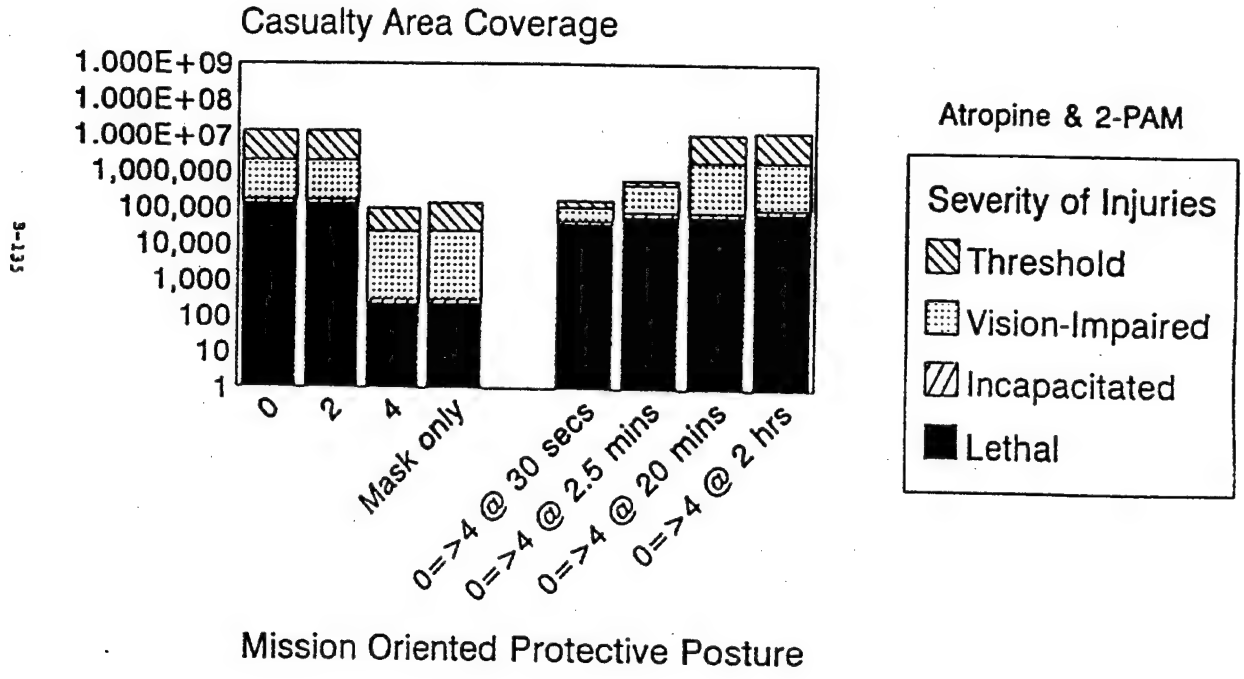
49°C (120°F), 6m/sec, stability B

Thirty-Two 250-kg Bombs
Sarin (GB)



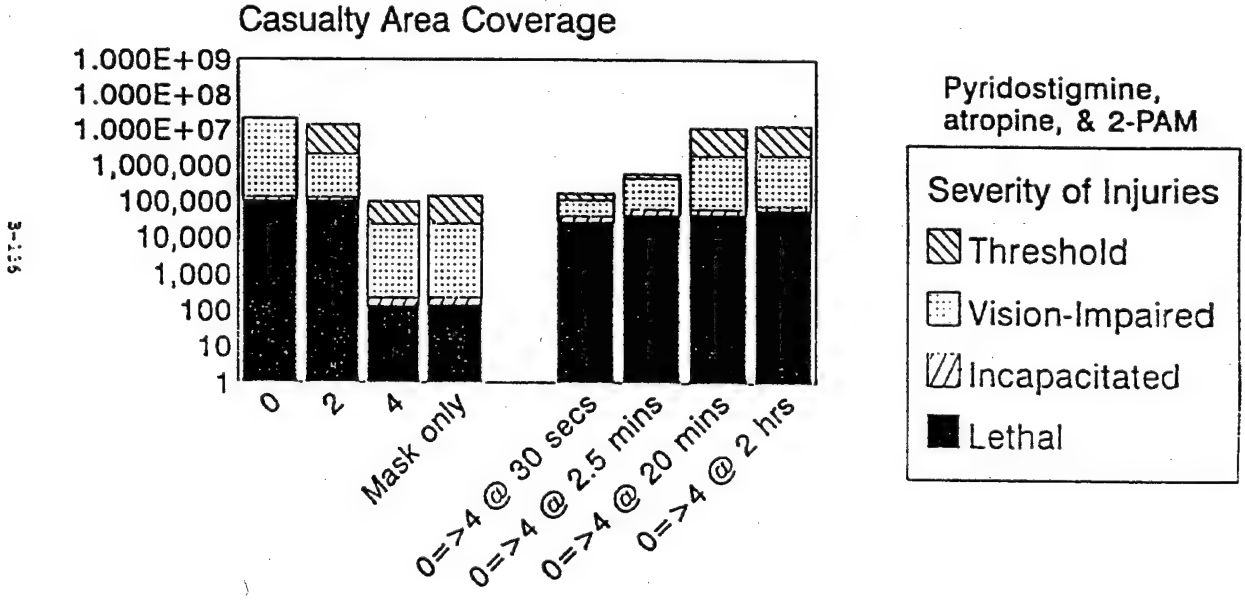
49°C (120°F), 6m/sec, Stability B

Thirty-Two 250-kg Bombs
Sarin (GB)



49°C (120°F), 6m/sec, Stability B

Thirty-Two 250-kg Bombs
Sarin (GB)



49°C (120°F), 6m/sec, Stability B

BOMB

Mustard (HD)

Bomb - Mustard (HD)

An aircraft bombing attack consisting of 96 100 kilogram ground burst bombs was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. Each bomb contained slightly less than 30 kilograms of mustard. The fireplan was based on a stick release of 24 bombs on each of 4 aircraft flying nearly the same approach over the target. This attack could also be used to represent an attack by 1 bomber aircraft.

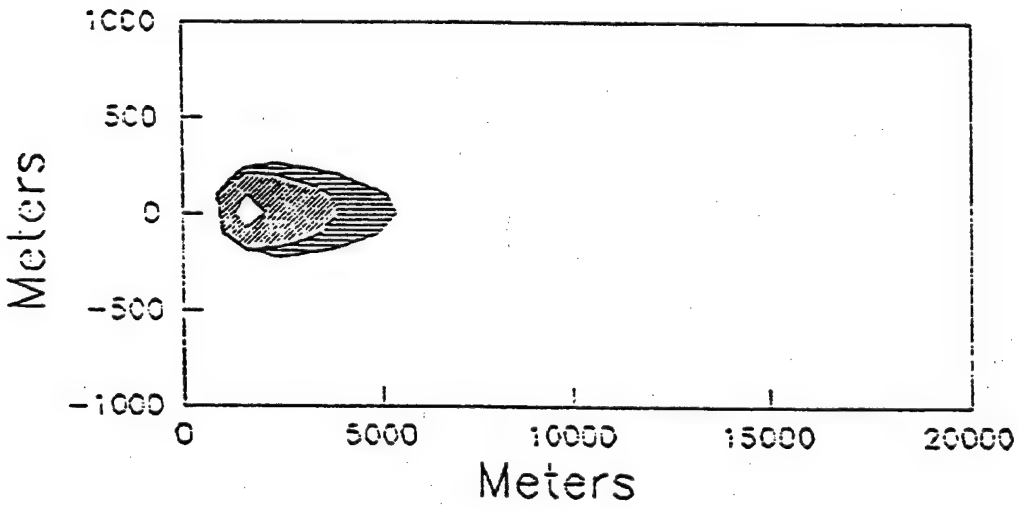
The peak liquid deposition from the attacks is very high in the range of 10 to 100 grams/square meter with no liquid area coverage greater than 1 square kilometer under any of the three meteorological cases.

The concentration across the target area drops below significant levels between 1 hour and 16 hours for the low temperature, low windspeed case and the moderate temperature, moderate windspeed case and between 3 minutes and 1 hour for the high temperature, high windspeed case.

The Pasquill stability category E shows the highest observed dosages of larger than 10,000 milligrams-minutes/cubic meters and an area coverage of 1 square kilometer. The neutral condition of Pasquill stability category D is only able to achieve a peak dosage of just less than 10,000 milligram-minutes/cubic meter and a maximum area coverage of less than 1 square kilometers. Pasquill stability category B case shows slightly lower peak dosages (still in the range of 10,000 milligram-minutes/cubic meter and 1 square kilometer of area coverage.

The level of lethal effects is only 0.1 square kilometers when a protective mask is not worn or put on between 1 hour and 16 hours after the attack.

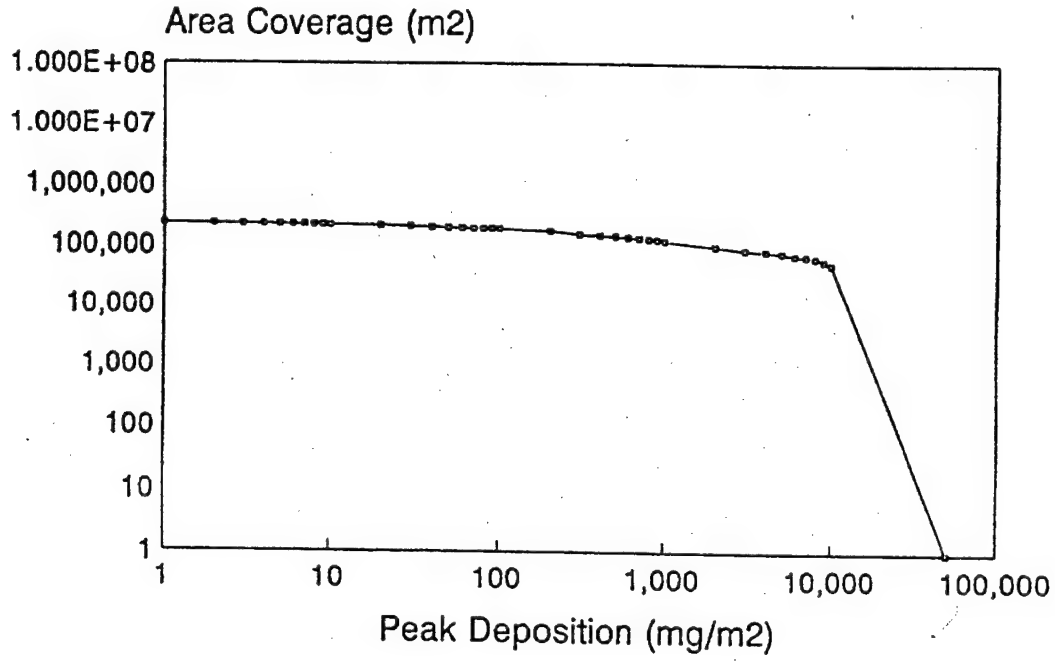
Ninety-Six 100-kg Bombs
Mustard (HD)



4oC (40oF)
1.5 m/sec
Stability E

Visually Impaired
Incapacitated
Lethal

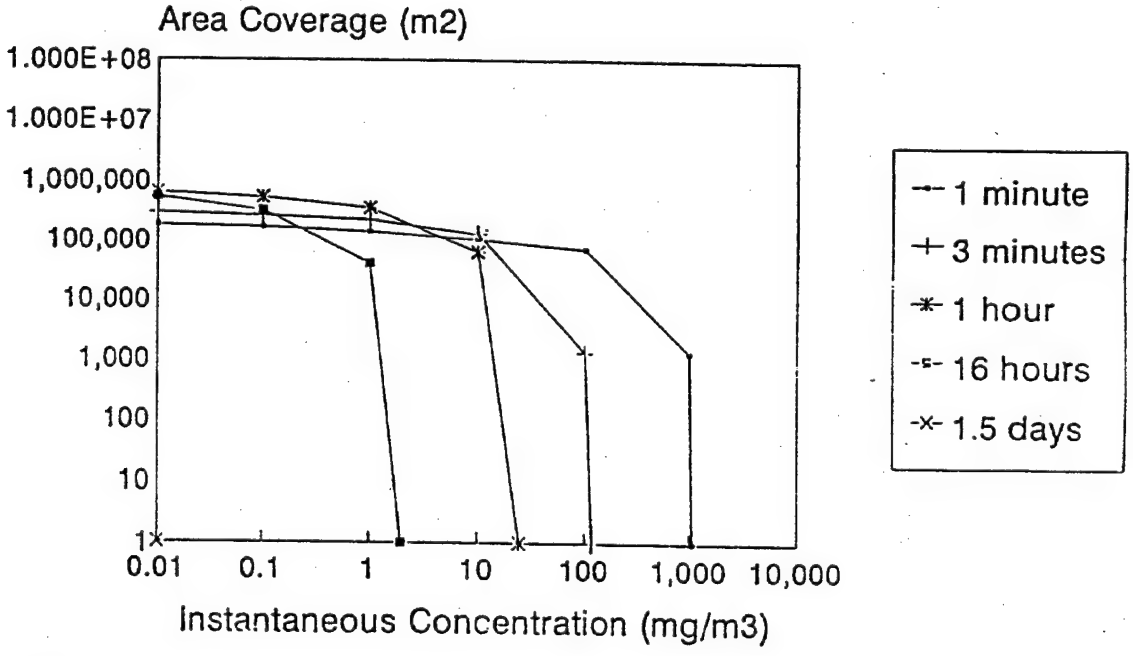
Ninety-Six 100-kg Bombs
Mustard (HD)



3-219

4°C (40°F), 1.5m/sec, stability E

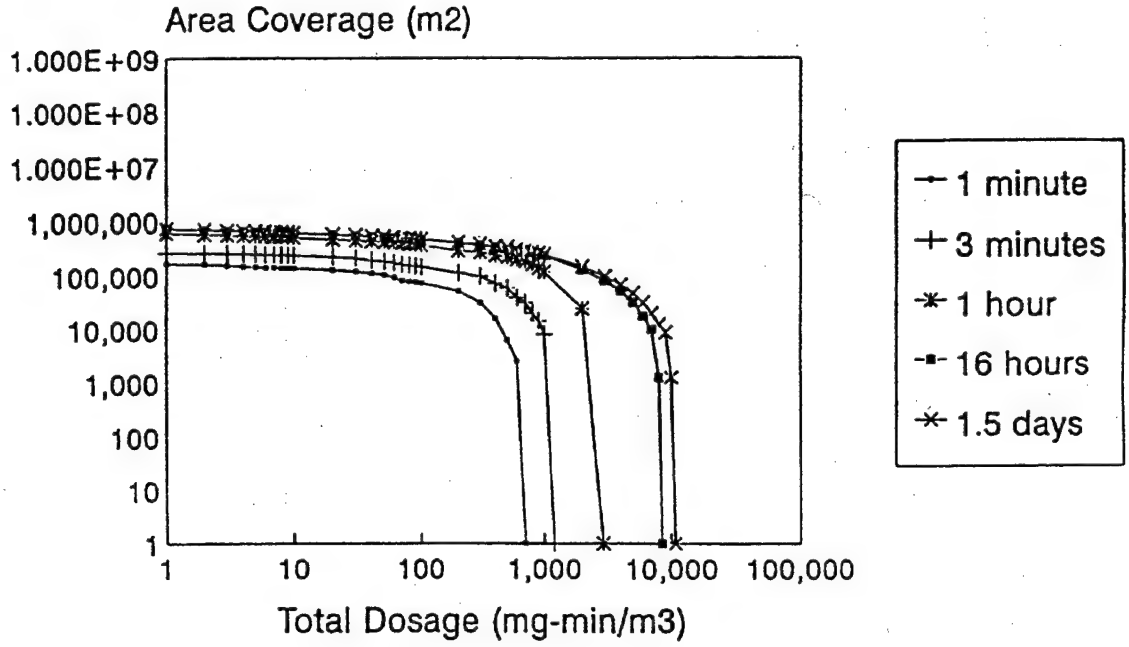
Ninety-Six 100-kg Bombs
Mustard (HD)



3-220

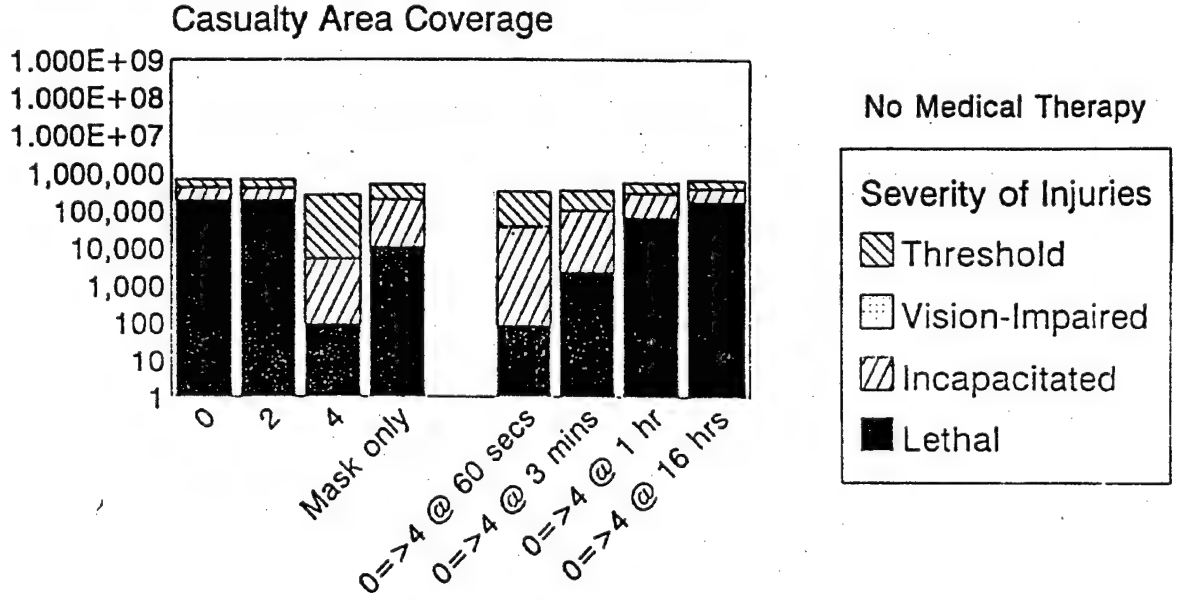
4°C (40°F), 1.5m/sec, stability E

Ninety-Six 100-kg Bombs
Mustard (HD)



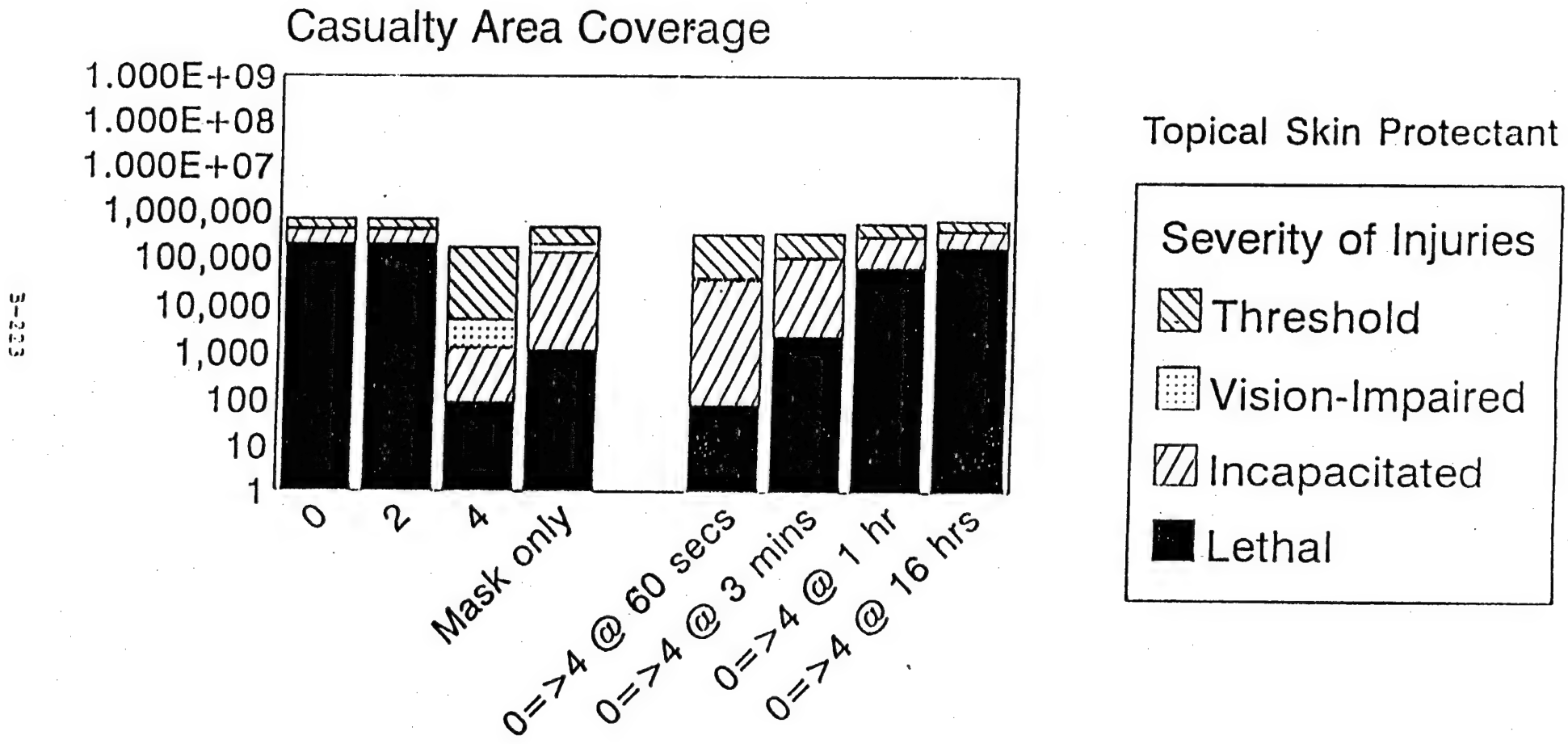
4°C (40°F), 1.5m/sec, stability E

Ninety-Six 100-kg Bombs
Mustard (HD)



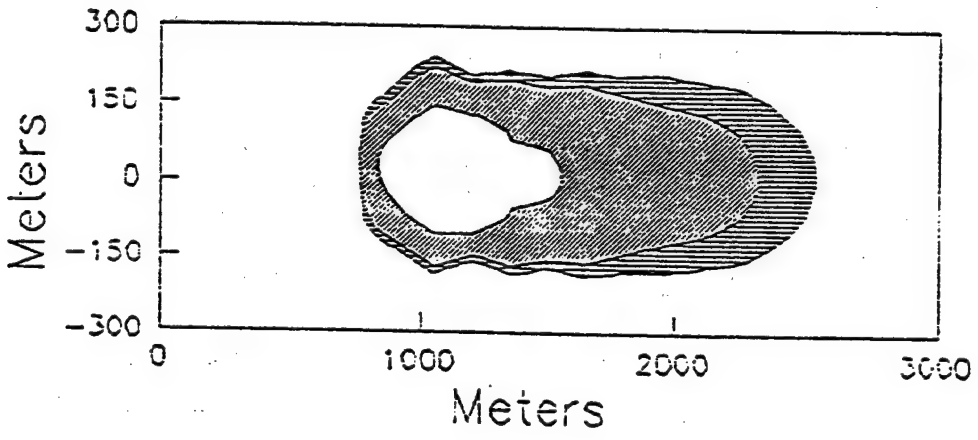
4°C (40°F), 1.5m/sec, stability E

Ninety-Six 100-kg Bombs
Mustard (HD)



4°C (40°F), 1.5m/sec, Stability E

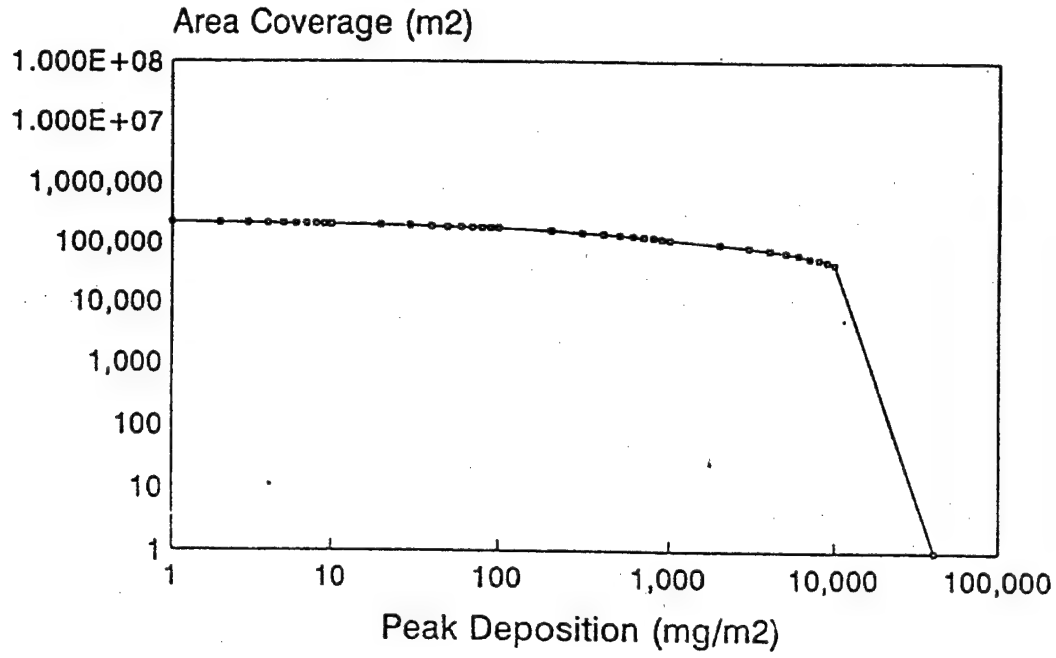
Ninety-Six 100-kg Bombs
Mustard (HD)



25oC (77oF)
3 m/sec
Stability D

Visually Impaired
Incapacitated
Lethal

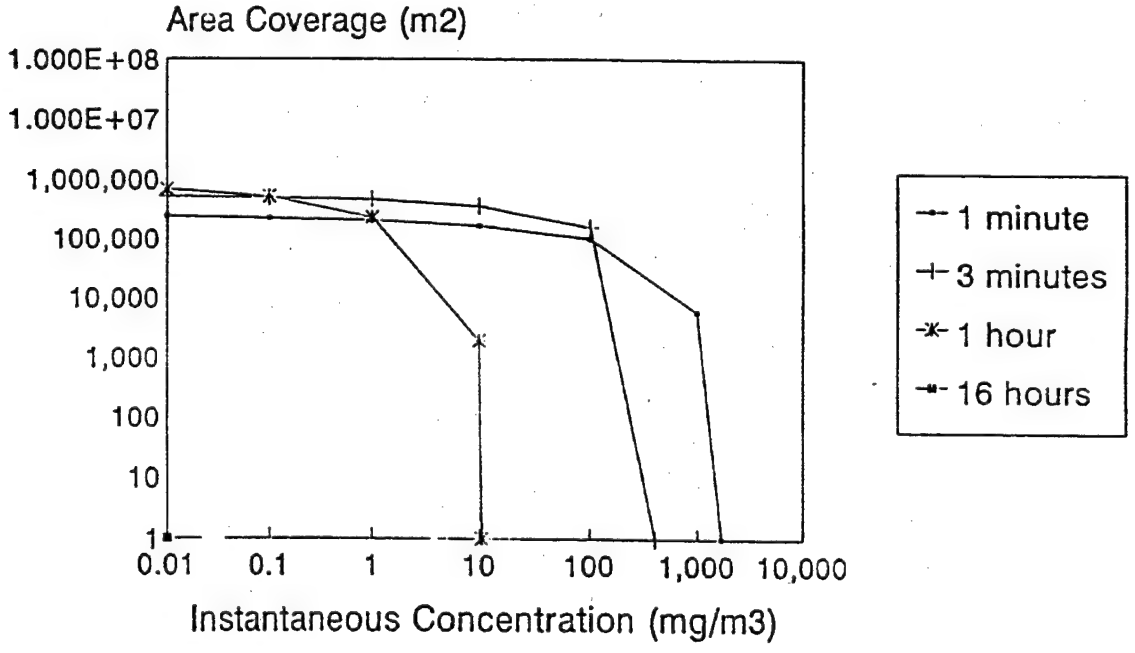
Ninety-Six 100-kg Bombs
Mustard (HD)



B-225

25°C (77°F), 3m/sec, stability D

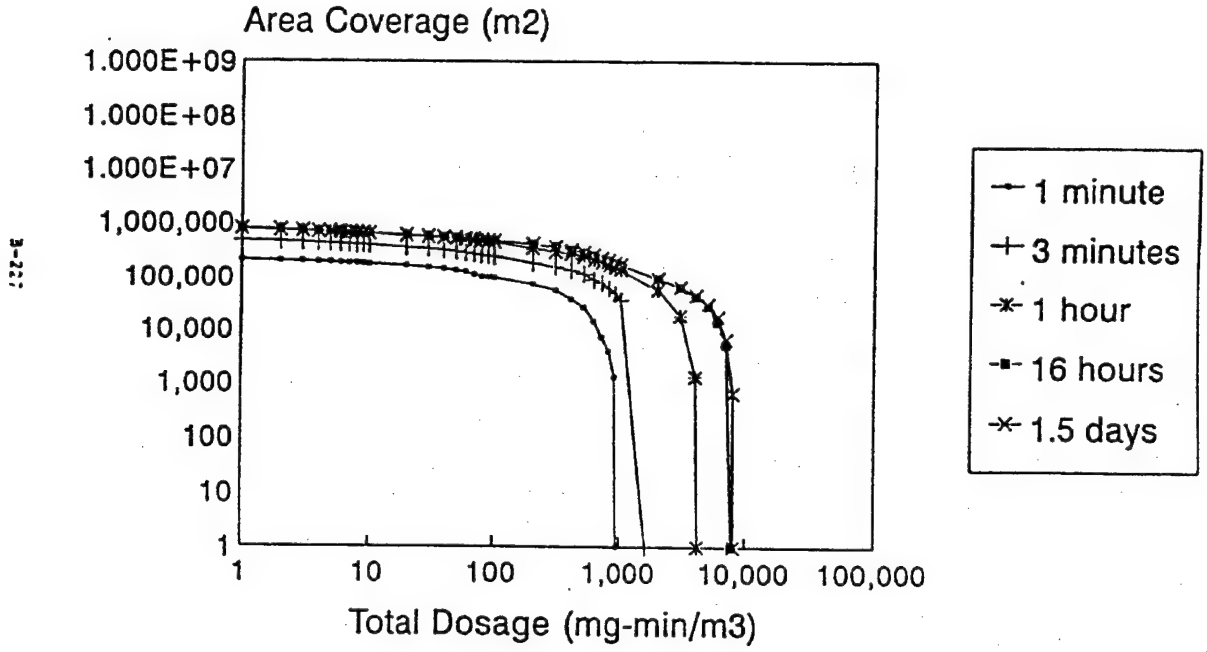
Ninety-Six 100-kg Bombs
Mustard (HD)



B-226

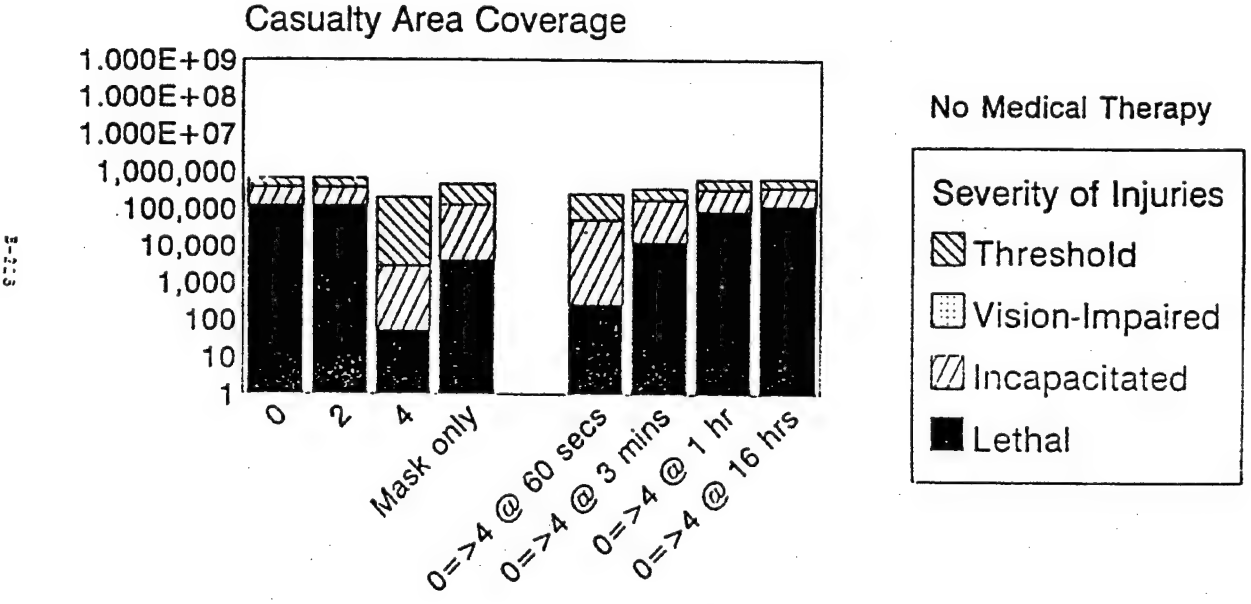
25°C (77°F), 3m/sec, stability D

Ninety-Six 100-kg Bombs
Mustard (HD)



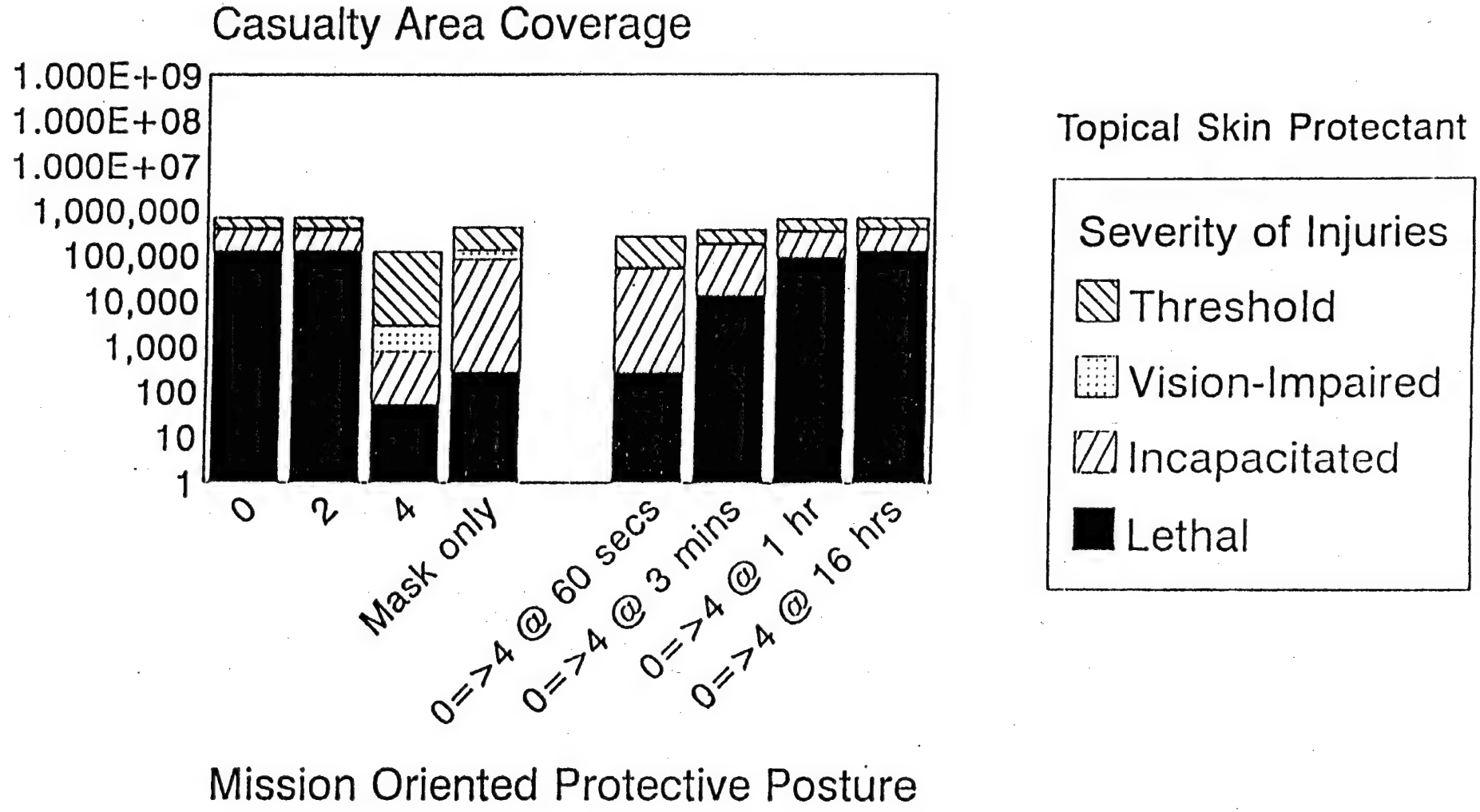
25°C (77°F), 3m/sec, stability D

Ninety-Six 100-kg Bombs
Mustard (HD)



25°C (77°F), 3m/sec, Stability D

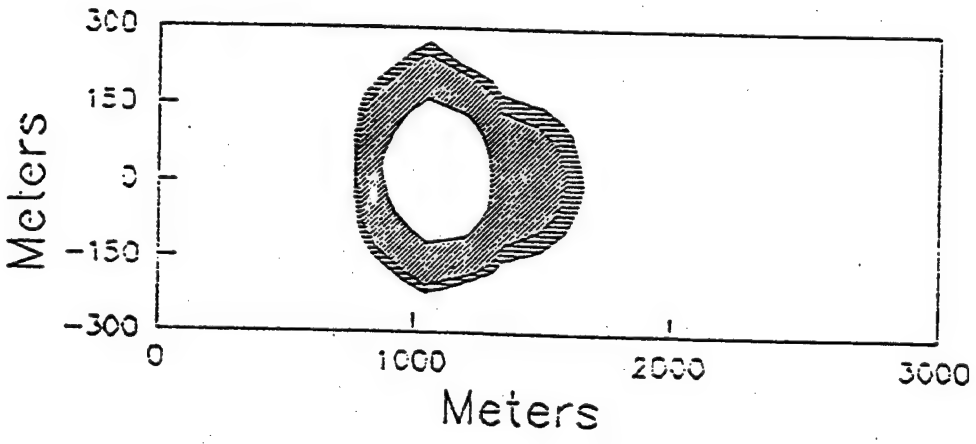
Ninety-Six 100-kg Bombs
Mustard (HD)



3-223

25°C (77°F), 3m/sec, Stability D

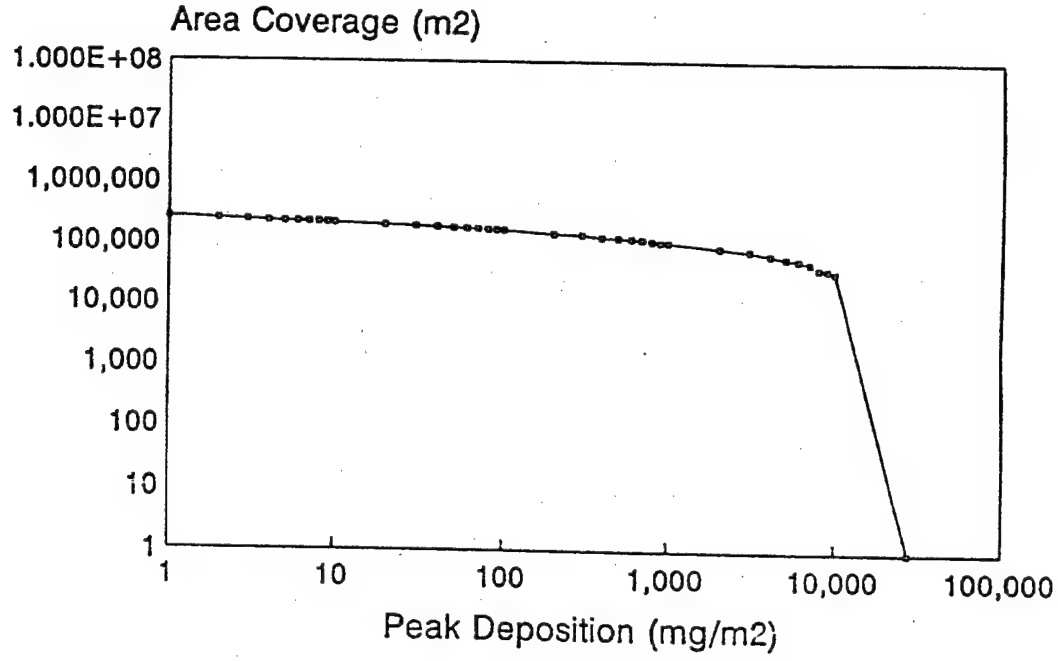
Ninety-Six 100-kg Bombs
Mustard (HD)



49oC (120oF)
6 m/sec
Stability B

Visually Impaired
Incapacitated
Lethal

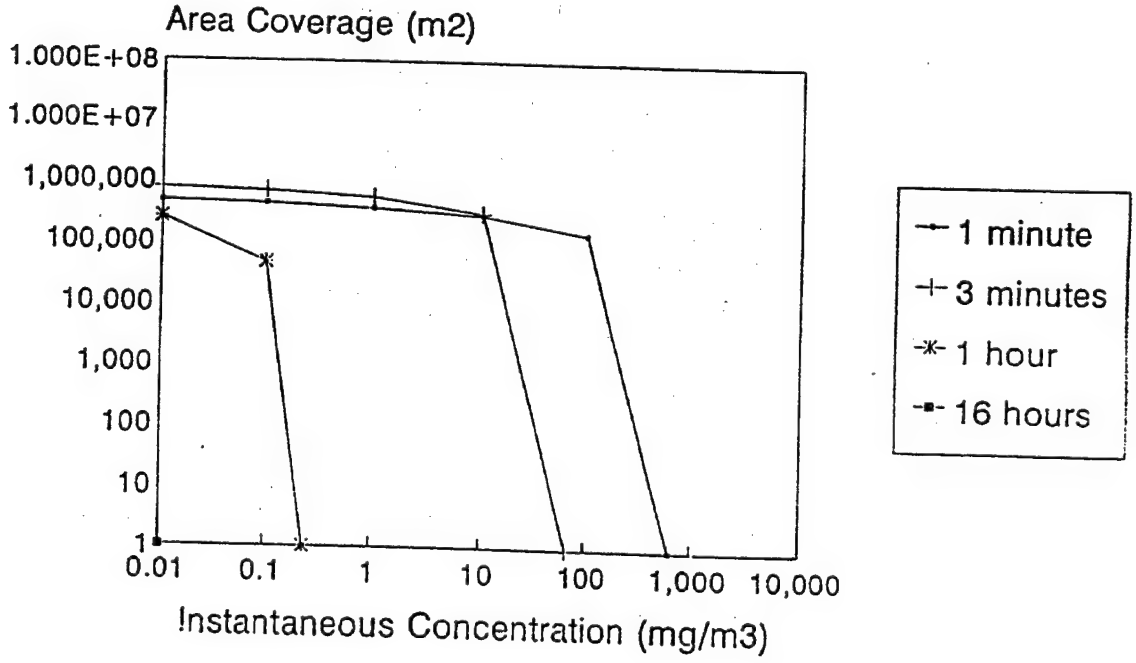
Ninety-Six 100-kg Bombs
Mustard (HD)



B-231

49°C (120°F), 6m/sec, stability B

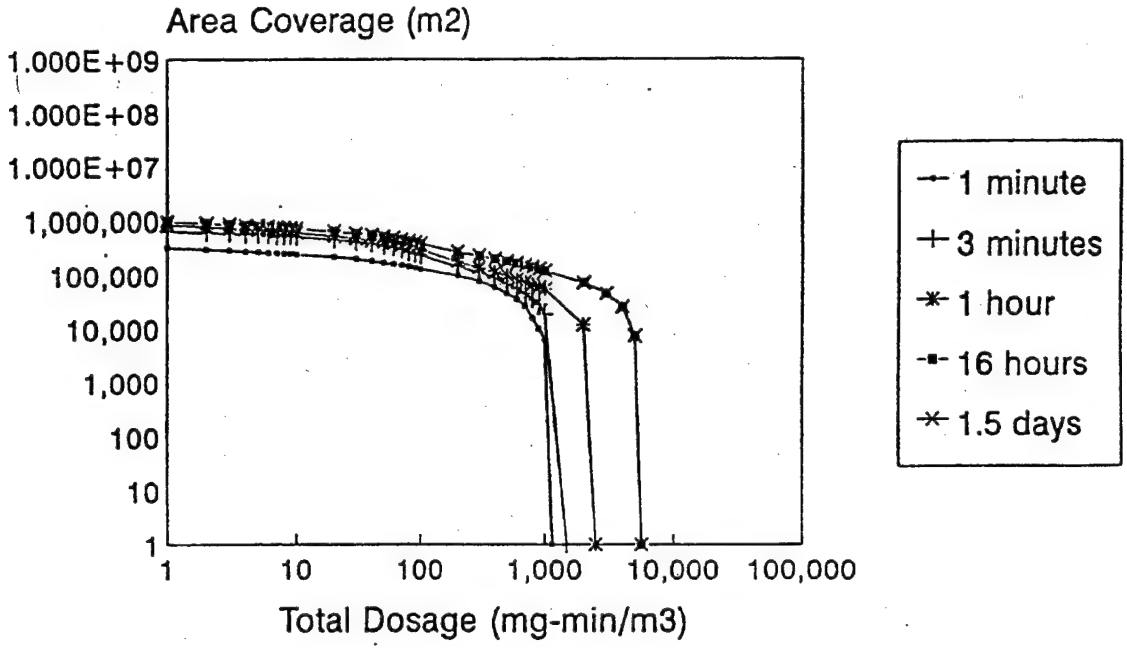
Ninety-Six 100-kg Bombs
Mustard (HD)



B-232

49°C (120°F), 6m/sec, stability B

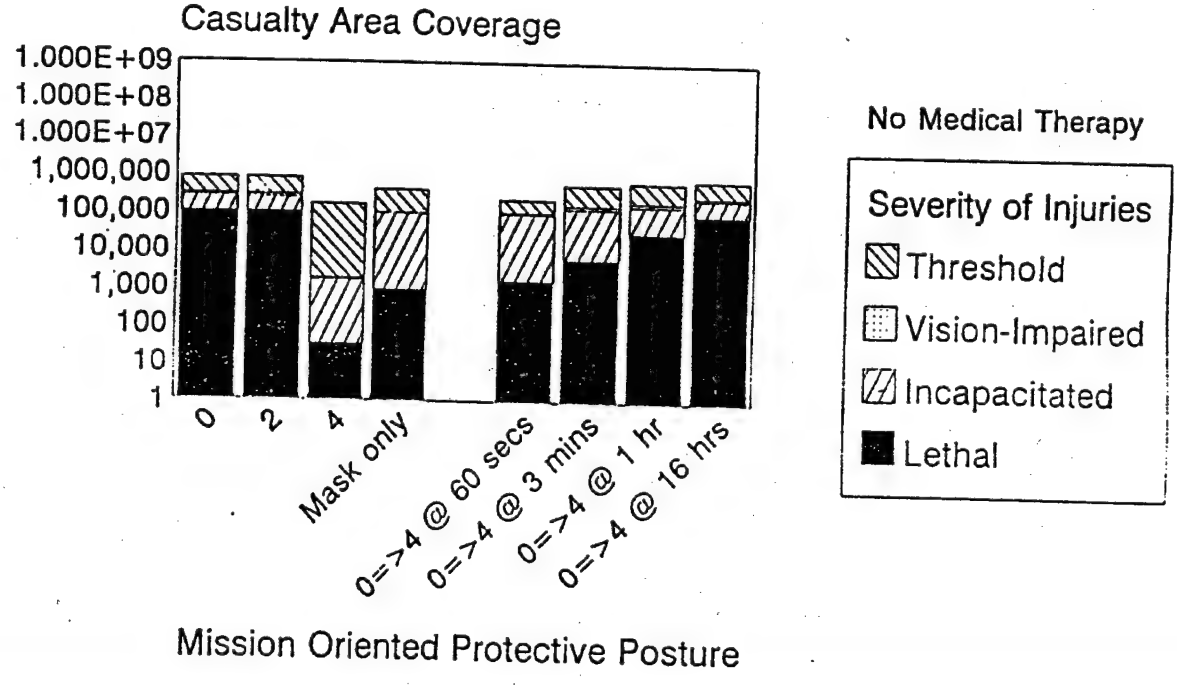
Ninety-Six 100-kg Bombs
Mustard (HD)



3-233

49°C (120°F), 6m/sec, stability B

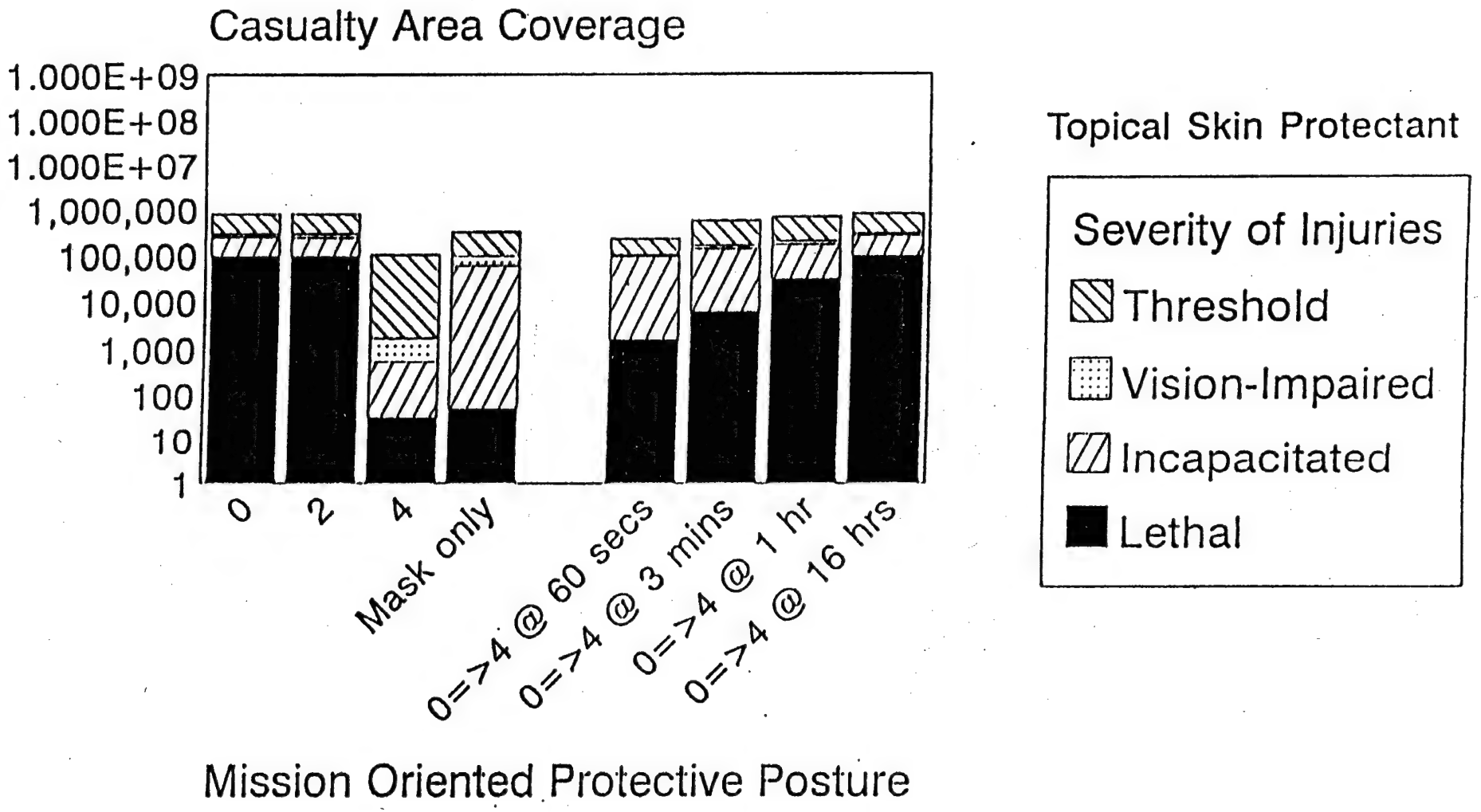
Ninety-Six 100-kg Bombs
Mustard (HD)



3-234

49°C (120°F), 6m/sec, Stability B

Ninety-Six 100-kg Bombs
Mustard (HD)



49°C (120°F), 6m/sec, Stability B

BOMB

Thickened Soman (TGD)

Bomb - Thickened Soman (TGD)

An aircraft bombing attack consisting of 32 250 kilogram air burst bombs was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. Each bomb contained slightly less than 50 kilograms of thickened soman. The fireplan was based on a stick release of 8 bombs on each of 4 aircraft flying nearly the same approach over the target. This attack could also be used to represent an attack by 1 bomber aircraft. The airburst bombs functions almost as a "flying spray tank" after released from the aircraft. The thickened agent forms larger relative drop sizes than explosive bombs typically used for ground burst. The larger drop sizes enable the agent to reach the ground reducing the evaporative flux from the droplet. The airburst allows the liquid to be spread over a larger area of the ground. Each munition was represented as producing an inclined line of agent with a mass median droplet diameter of approximately 2,500 microns.

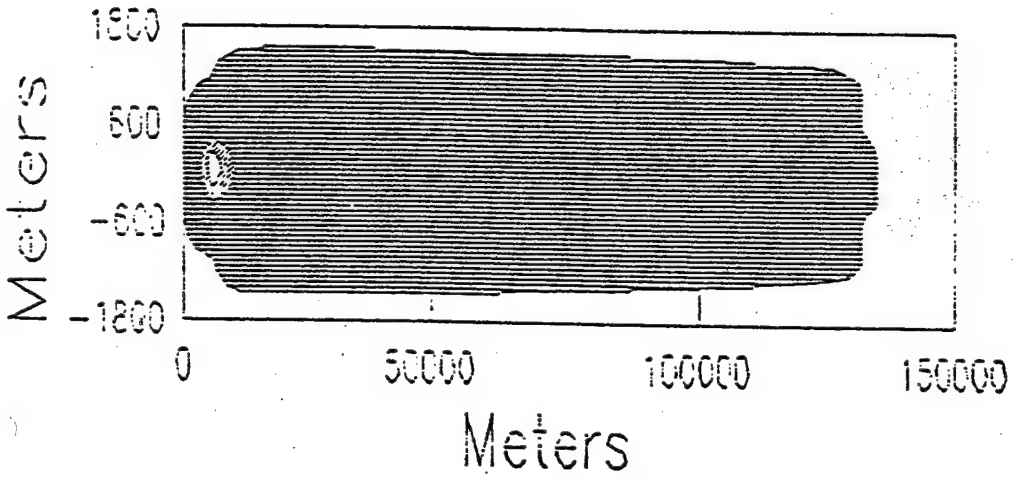
The peak liquid deposition from the attacks is very high in the range of 10 to 100 grams/square meter with no liquid area coverage greater than 1 square kilometer under any of the three meteorological cases.

The concentration across the target area drops below significant levels after 16 hours for the low temperature, low windspeed case and between 20 minutes and 2 hour for both the moderate temperature, moderate windspeed case and the high temperature, high windspeed case.

The inversion condition of Pasquill stability category E shows the highest observed dosages reaching levels approaching 10,000 milligram-minutes/cubic meters. Also significant is that this case results in agent dosage being carried to almost 100 square kilometers. While the neutral condition of Pasquill stability category D is only able to achieve a peak dosage of just slightly lower than the inversion condition, the maximum area coverage of less is nearly identical at almost 100 square kilometers. The Pasquill atmospheric stability category B shows even lower peak dosages and area coverages.

The casualty area coverage charts show a drop of more than an order of magnitude in lethal level effects as the temperature go from cold to hot. There is a two to three order of magnitude reduction in lethal area for wearing the protective mask. There is only a small improvement in lethal area afforded by adding the ensemble (MOPP 2 vs MOPP 0 or MOPP4 vs Mask only). The lethal area coverage shows that donning the mask at 30 seconds after the attack results in nearly a 2 order of magnitude more area coverage than occurs when the mask is on at the time of attack. Medical intervention reduces the likely lethal area by almost an order of magnitude (more than a 90% reduction).

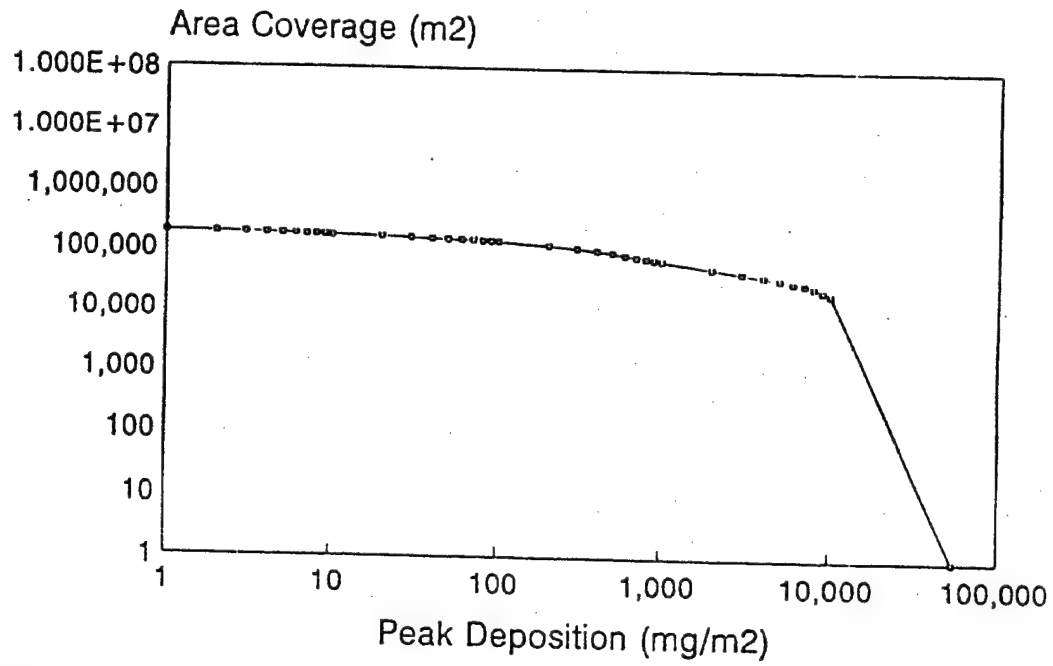
Thirty-Two 250-kg Airburst Bombs
Thickened Soman (GD)



4oC (40oF)
1.5 m/sec
Stability E

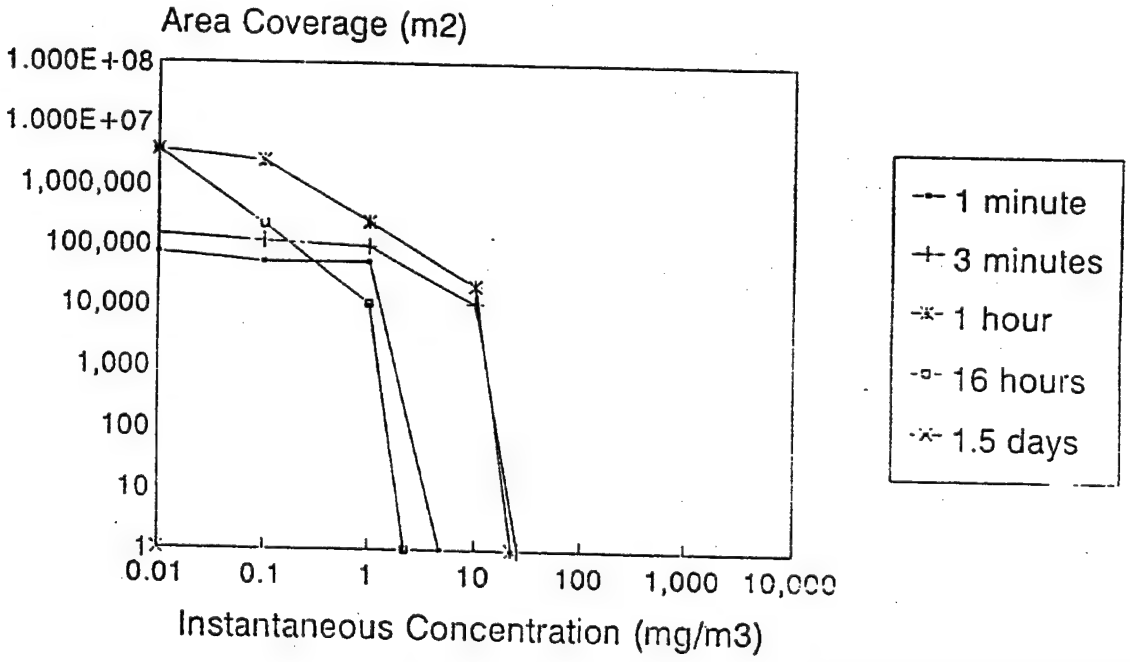
Visually Impaired
Incapacitated
Lethal

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



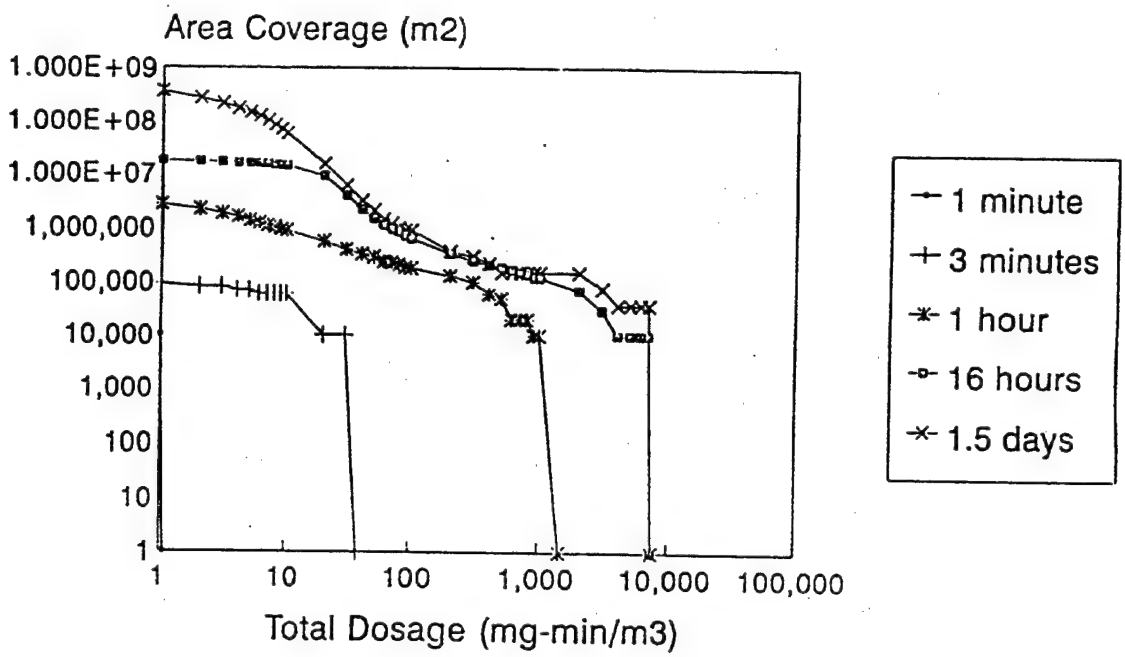
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



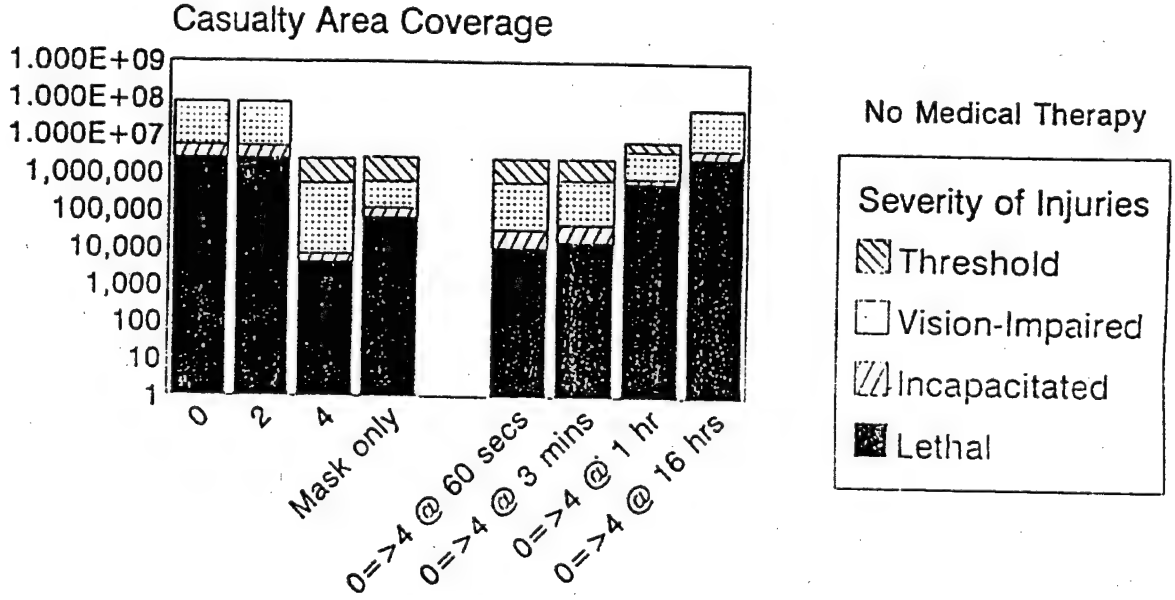
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



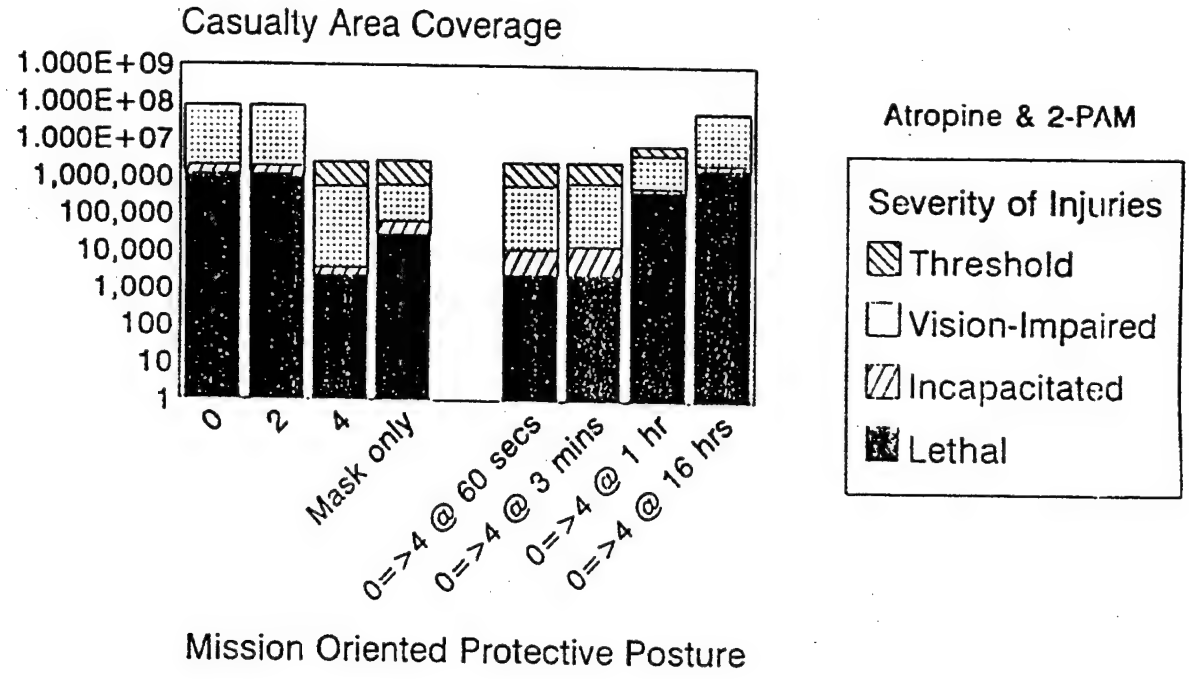
4°C (40°F), 1.5m/sec, stability E

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



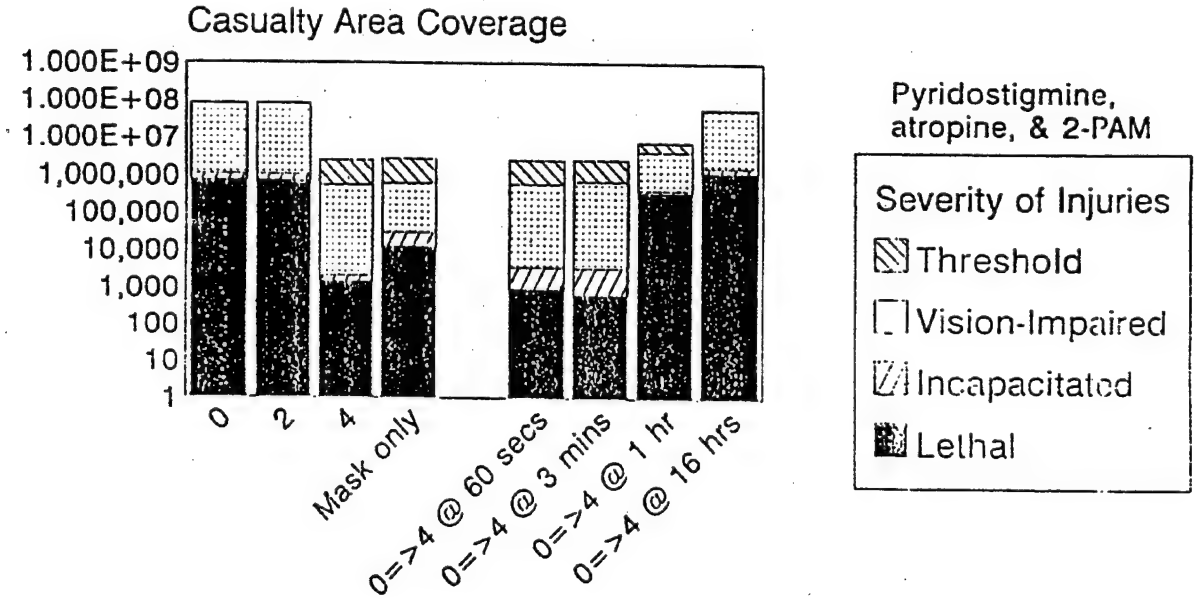
4°C (40°F), 1.5m/sec, Stability E

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



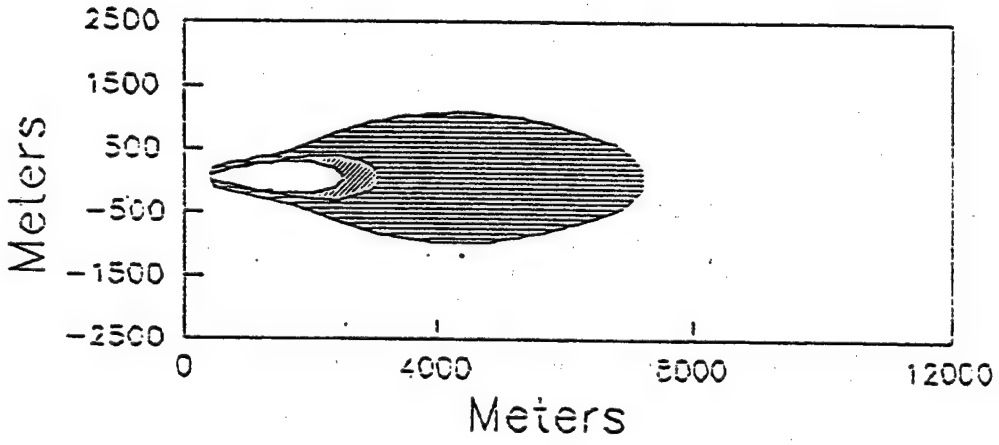
4°C (40°F), 1.5m/sec, Stability E

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



4°C (40°F), 1.5m/sec, Stability E

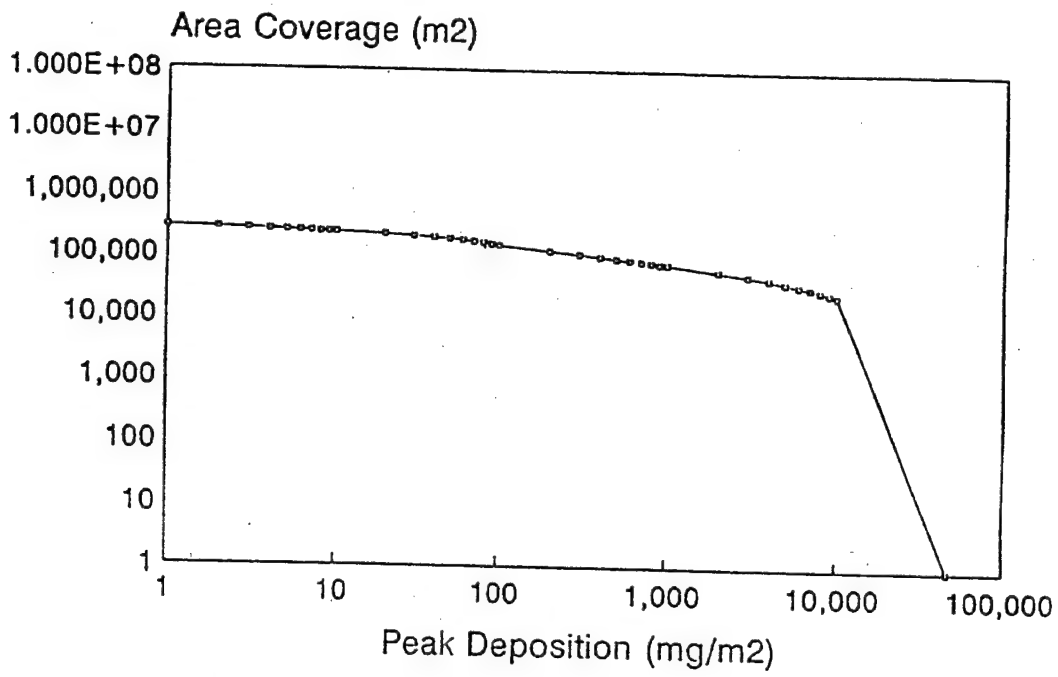
Thirty-Two 250-kg Airburst Bombs
Thickened Soman (GD)



25oC (77oF)
3 m/sec
Stability D

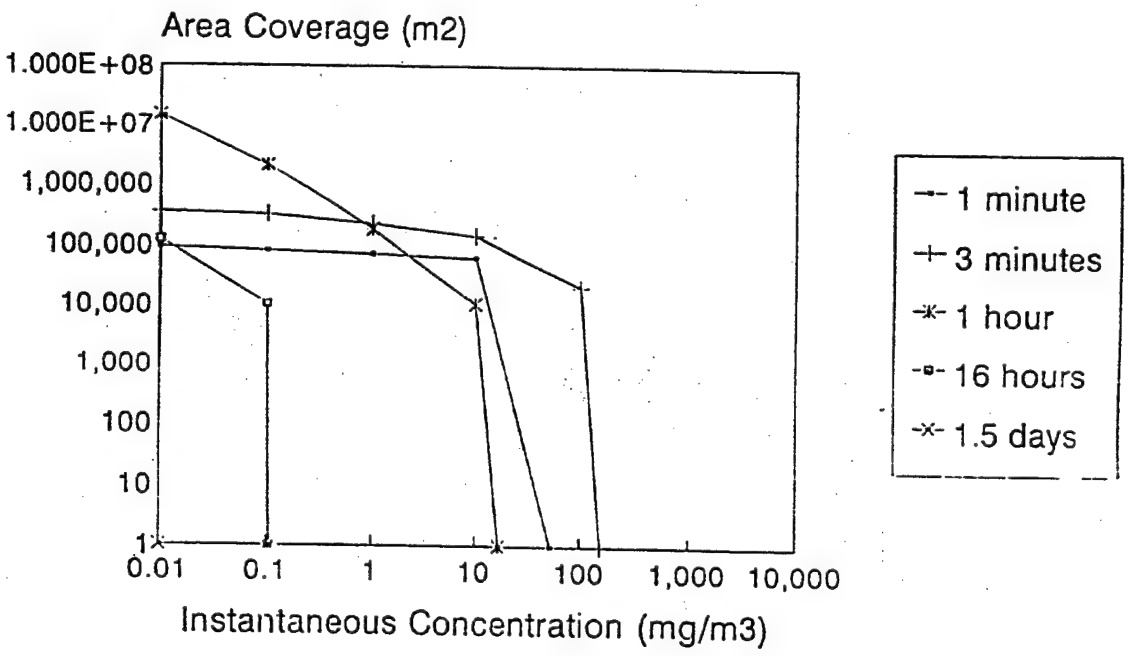
Visually Impaired
Incapacitated
Lethal

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



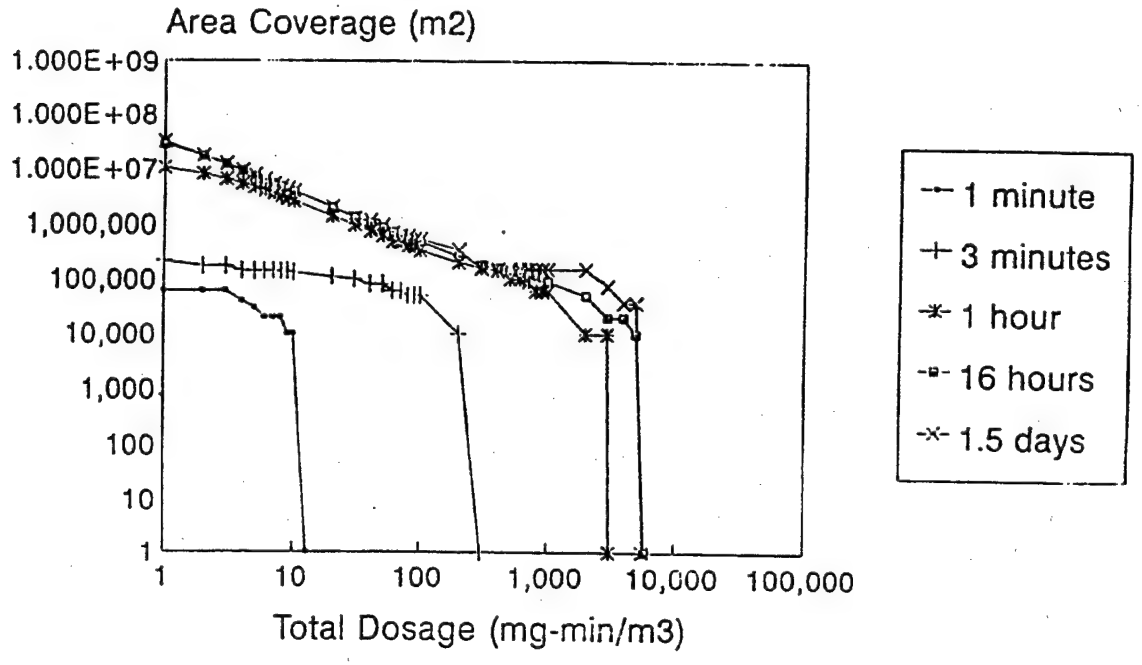
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



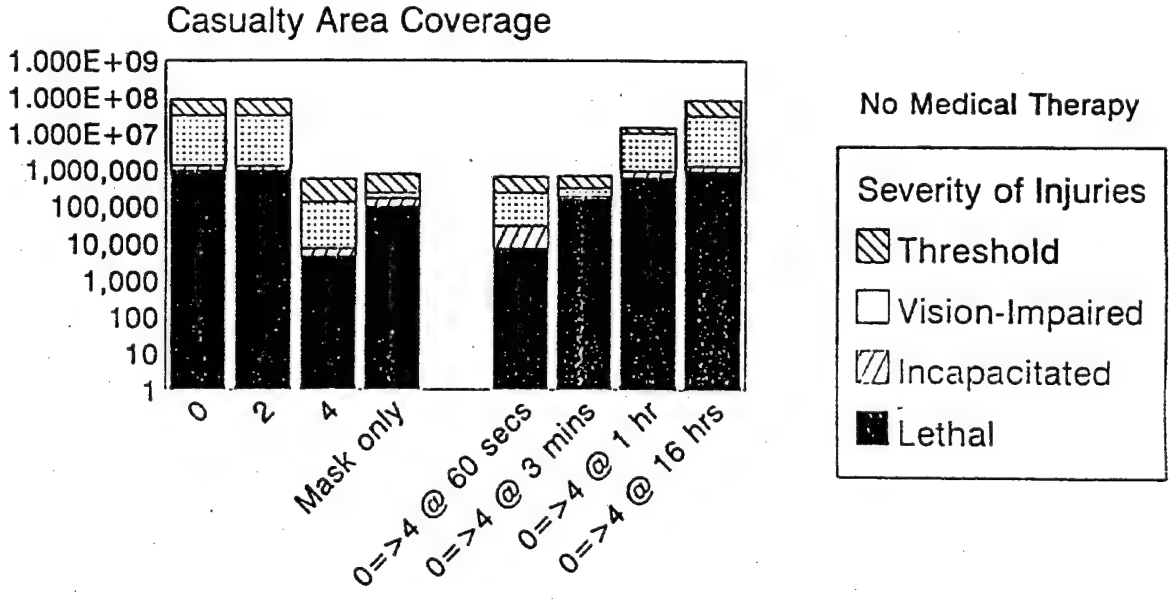
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



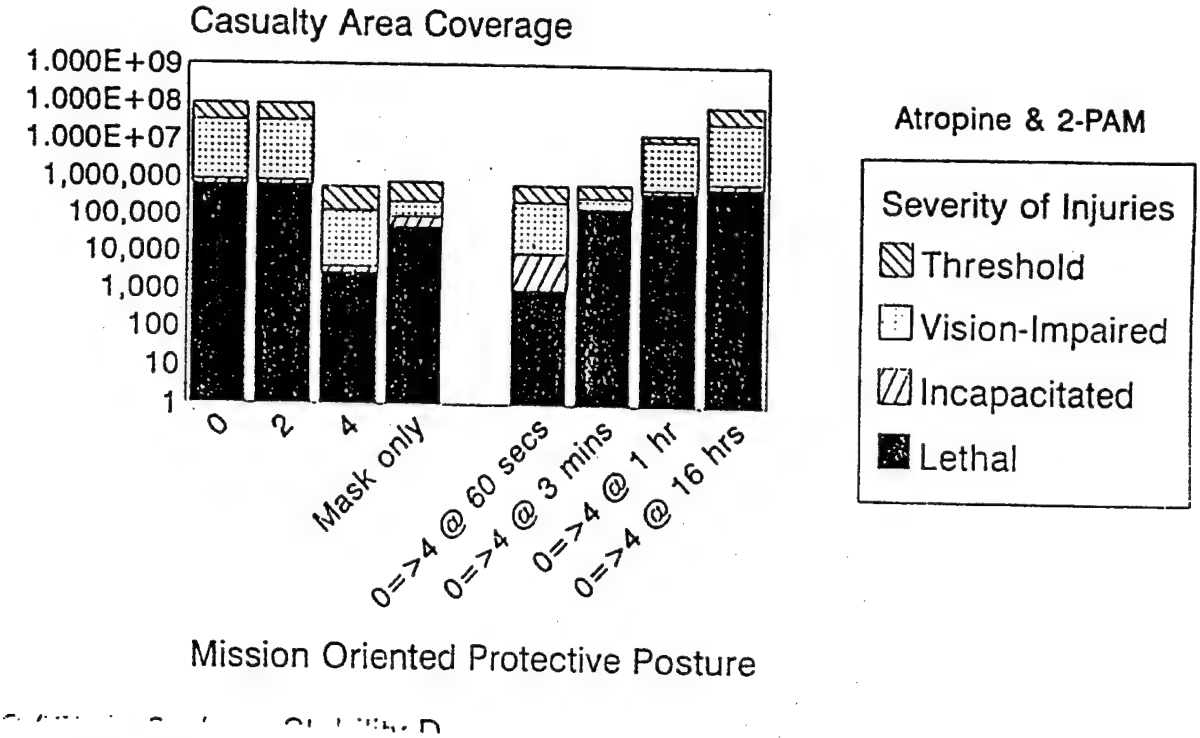
25°C (77°F), 3m/sec, stability D

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)

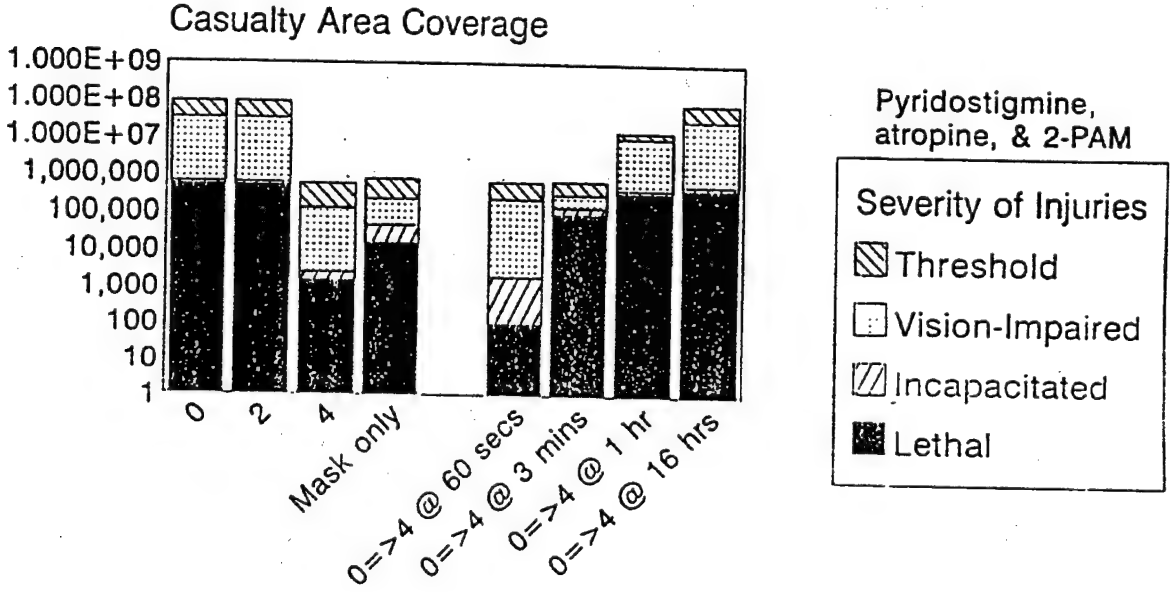


25°C (77°F), 3m/sec, Stability D

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)

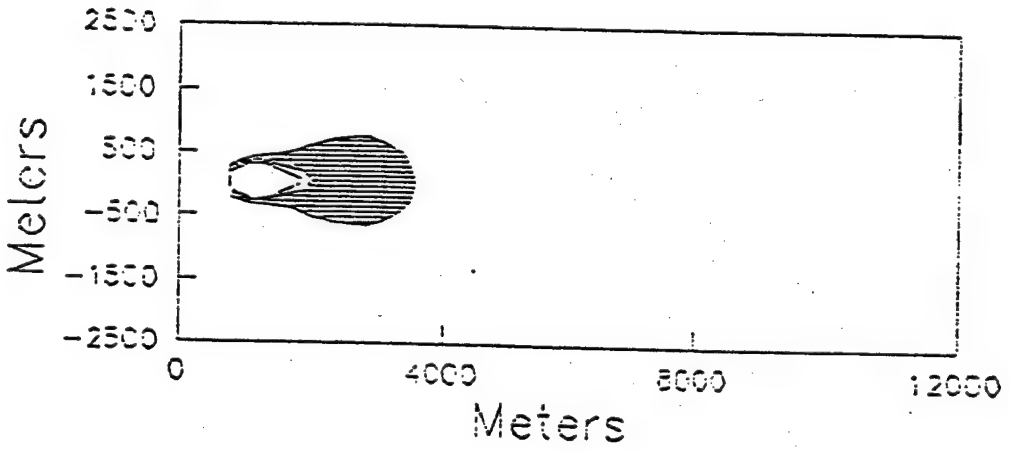


Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



25°C (77°F), 3m/sec, Stability D

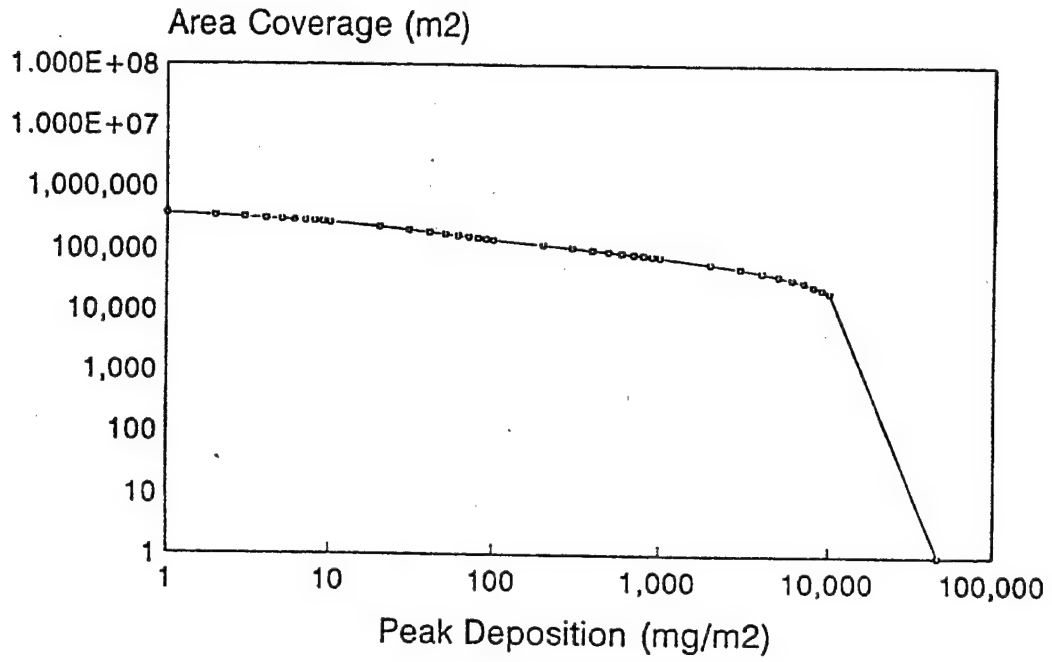
Thirty-Two 250-kg Airburst Bombs
Thickened Soman (GD)



49oC (120oF)
6 m/sec
Stability B

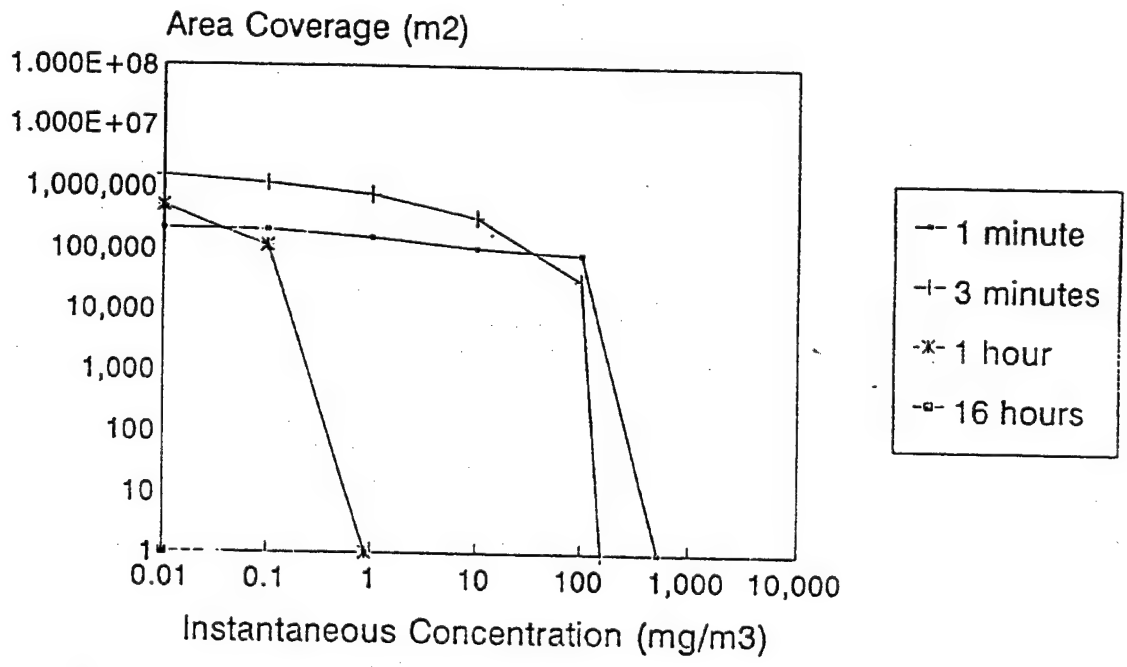
Visually Impaired
Incapacitated
Lethal

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



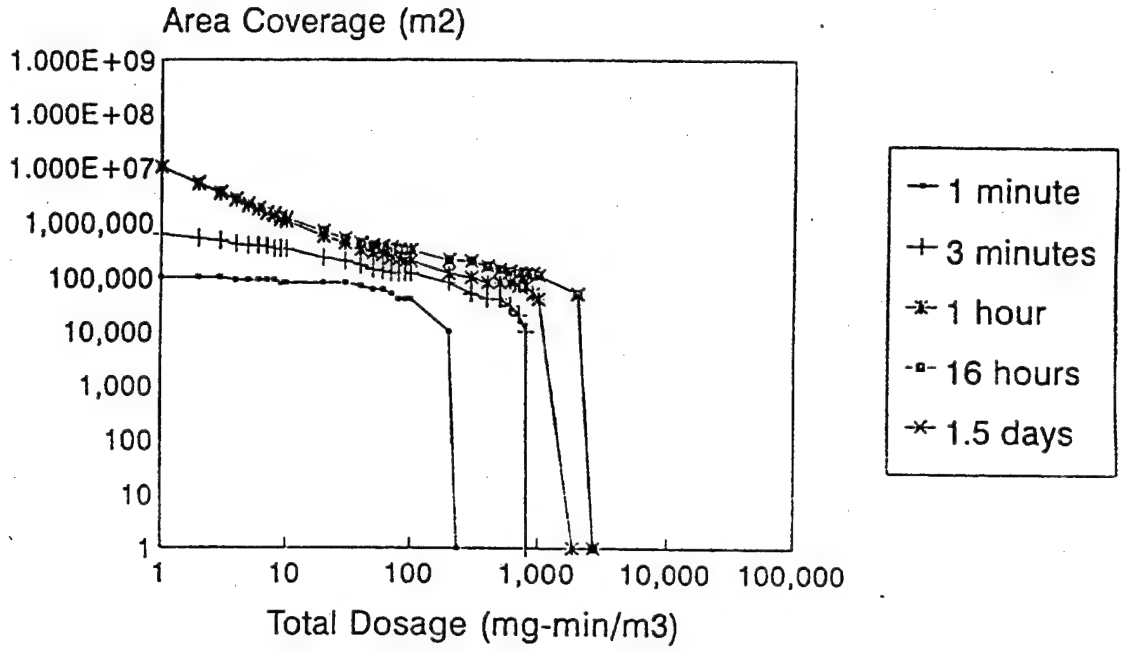
49°C (120°F), 6m/sec, stability B

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



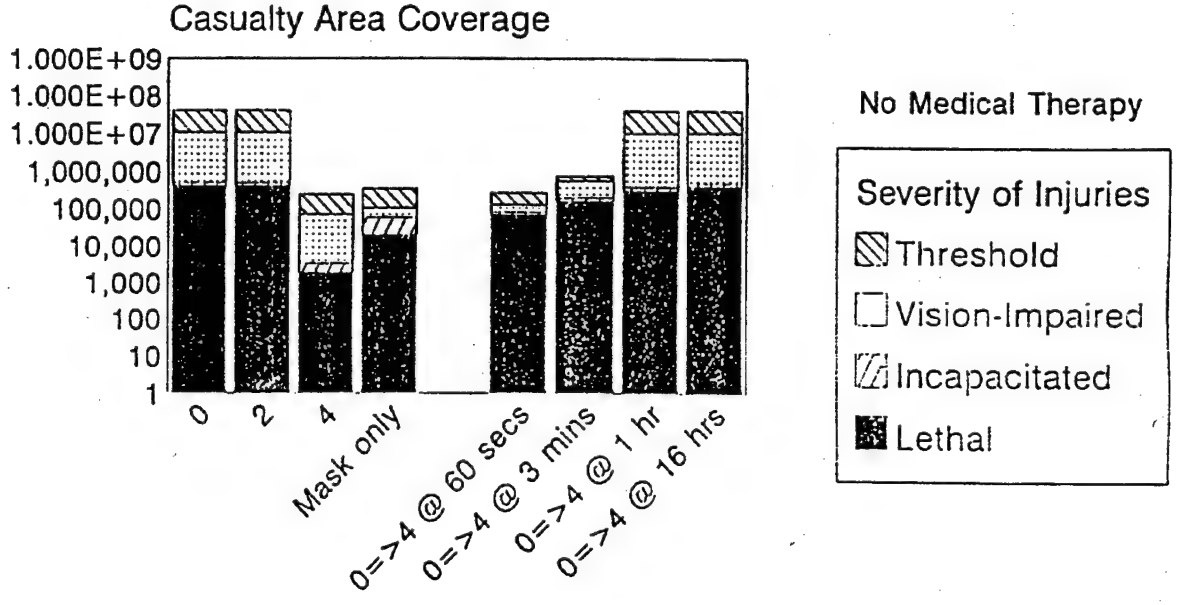
49°C (120°F), 6m/sec, stability B

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



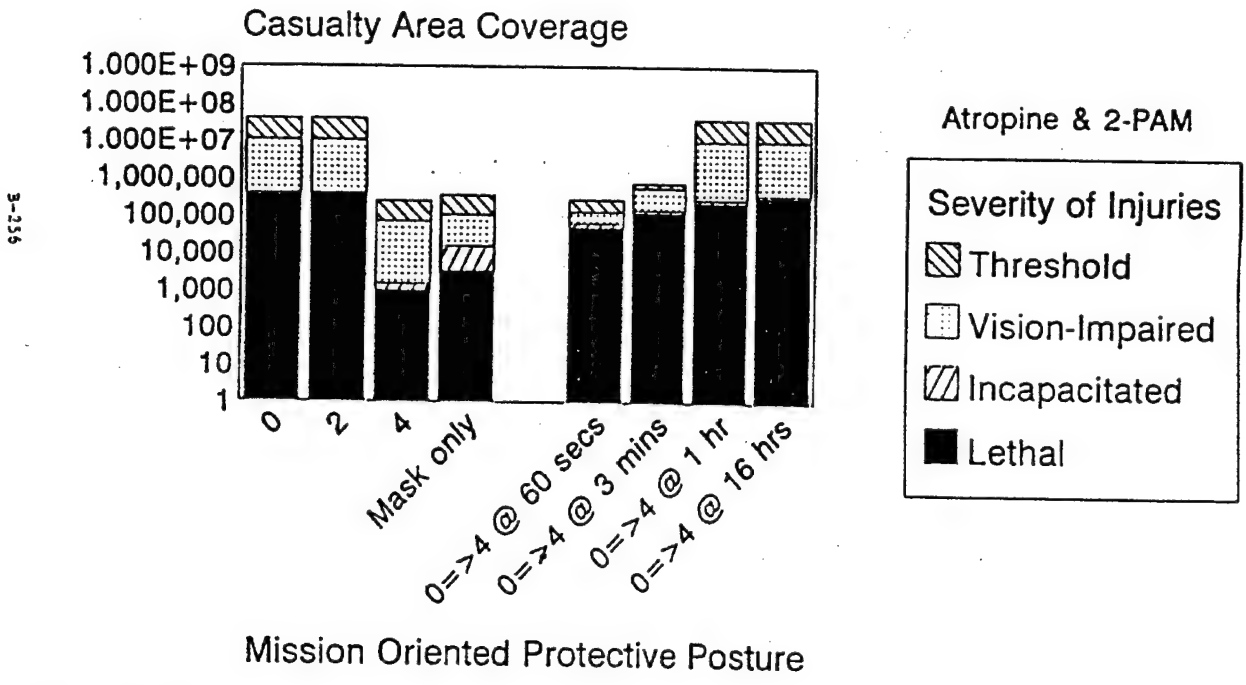
49°C (120°F), 6m/sec, stability B

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



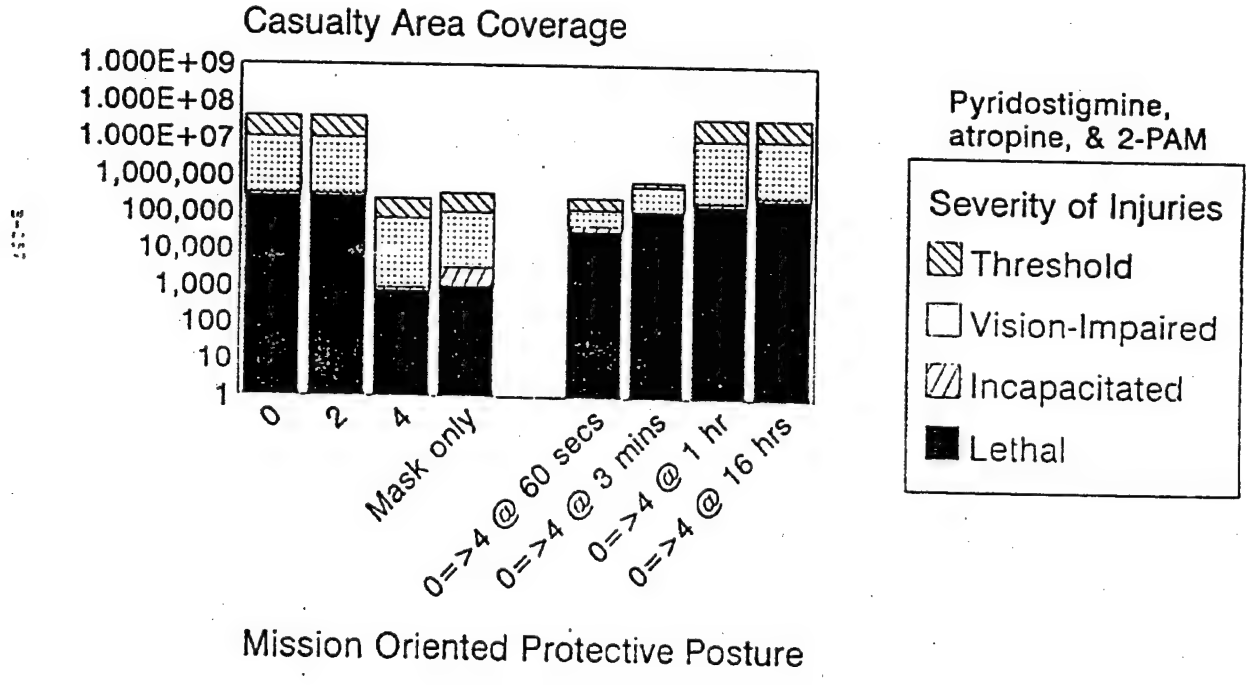
49°C (120°F), 6m/sec, Stability B

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



49°C (120°F), 6m/sec, Stability B

Thirty-Two 250-kg Airburst Bombs Thickened Soman (GD)



49°C (120°F), 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE WITH SUBMUNITIONS

Sarin (GB)

Tactical Ballistic Missile with Submunitions - Sarin (GB)

Approximately 100 submunitions, each containing just over 2 kilograms of sarin was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The submunitions were released from the tactical ballistic missile at an altitude of approximately 1.5 kilometers producing a 1.2 kilometer diameter submuniton pattern on the ground.

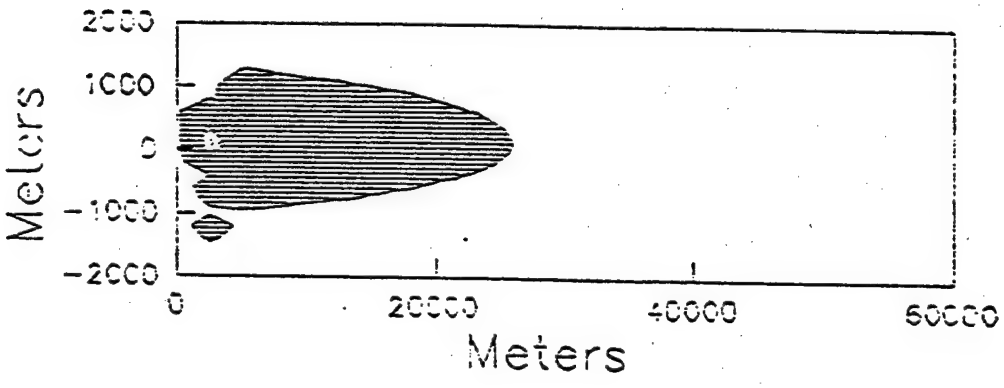
The peak liquid deposition from the attacks was between 1 and 10 grams/square meter with no liquid area coverage much higher than 0.1 square kilometer under all three of the meteorological cases.

Concentrations do not drop below significant levels for more than 2 hours and less than 1.5 days for the low temperature, low windspeed case; between 20 minutes and two hours in the moderate temperature, moderate windspeed case; and between 2.5 minutes and 20 minutes for the high temperature, high windspeed case.

The peak dosage is between 100 and 1,000 milligram-minutes/cubic meters for all three meteorology conditions. Maximum dosage area coverage is between 10 and 100 square kilometers for the low temperature, low windspeed and the moderate temperature, moderate windspeed cases. The peak dosage is just less than 10 square kilometers for the high temperature case.

Unprotected lethalities for the low temperature case was approximately 1 square kilometers while the moderate and high temperature cases yielded a likely area coverage of approximately 0.1 and 0.01 square kilometers respectively. There was more than a four order of magnitude reduction (more than a 99.99 per cent reduction) in likely casualty area coverage if the mask is worn, but only an order of magnitude (or a 90 per cent reduction) if masking occurs 1 minute after the start of the attack.

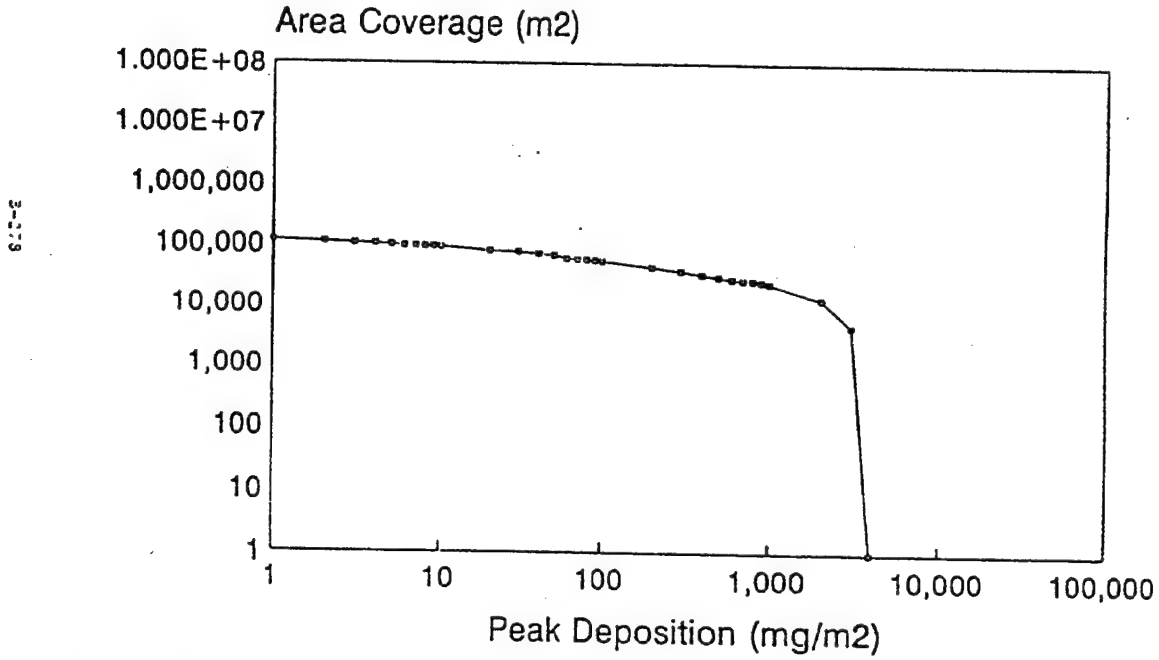
Tactical Ballistic Missile w/ Submunitions
Sarin (GB)



4oC (40oF)
1.5 m/sec
Stability E

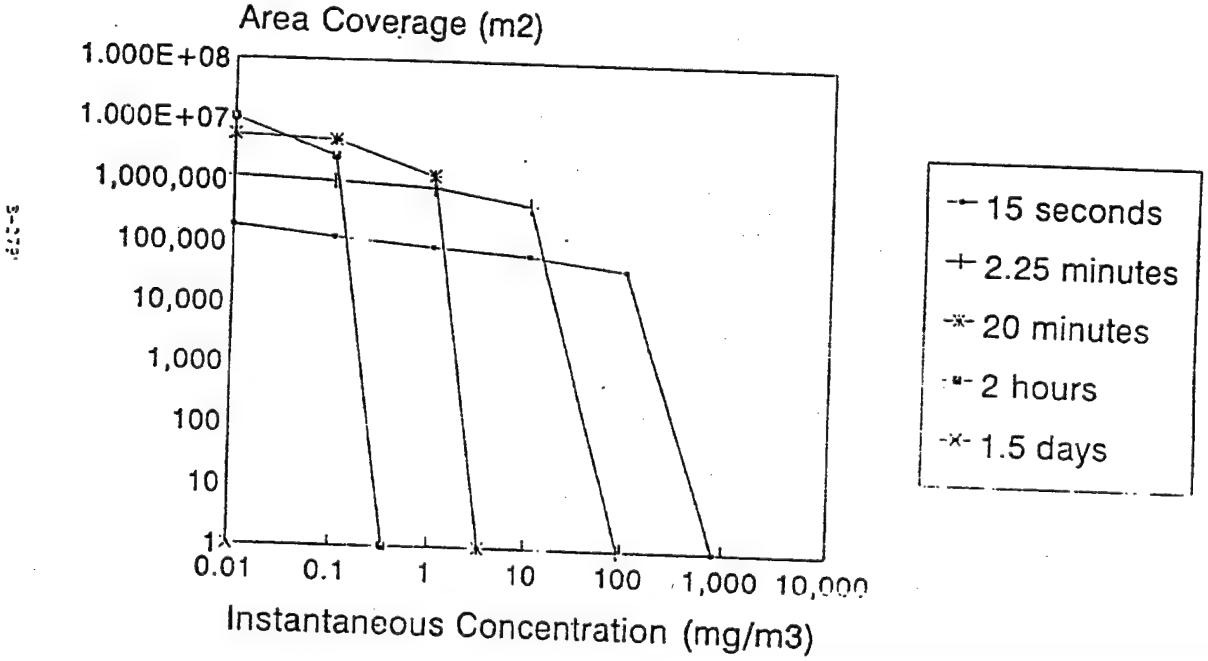
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile w/Submunitions Sarin (GB)



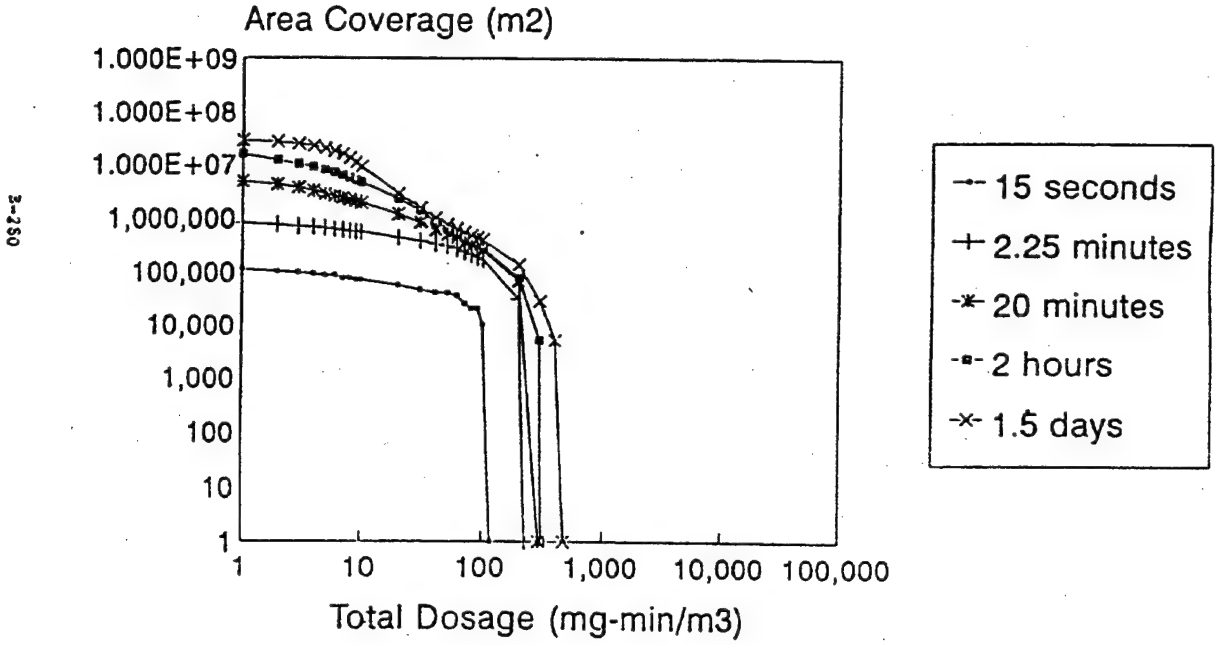
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile w/Submunitions Sarin (GB)



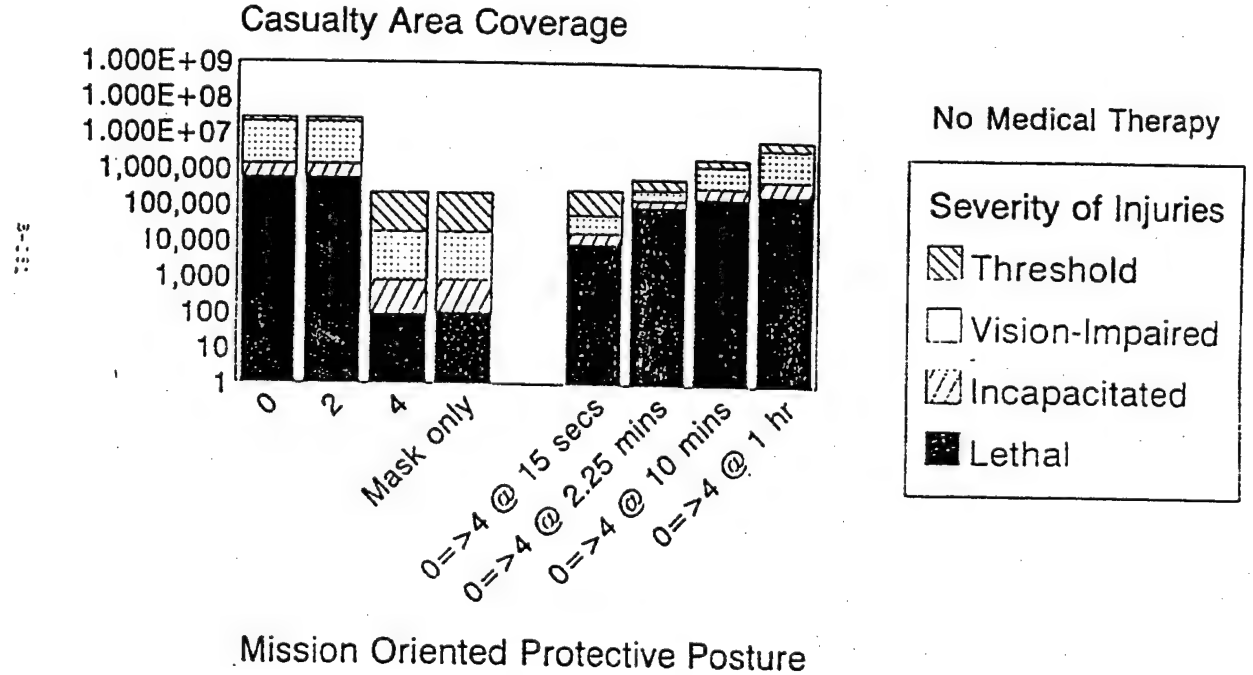
4°C (40°F). 1.5m/sec, stability E

Tactical Ballistic Missile w/Submunitions Sarin (GB)

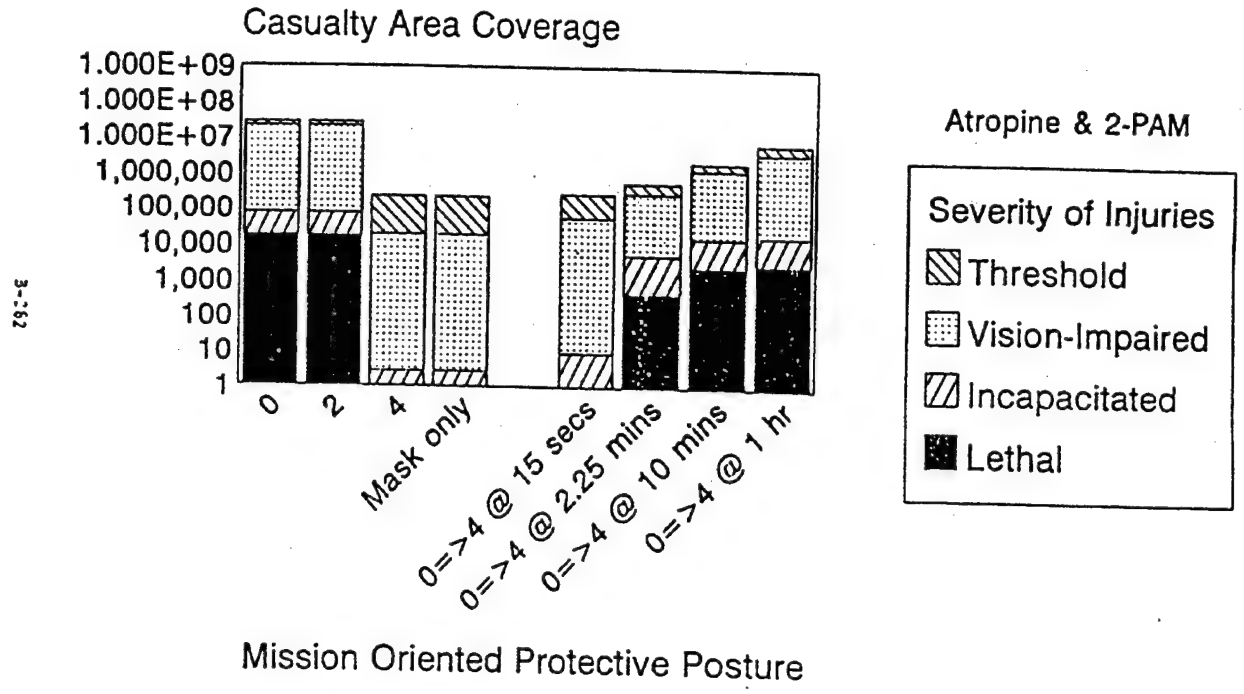


4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile with Submunitions Sarin (GB)

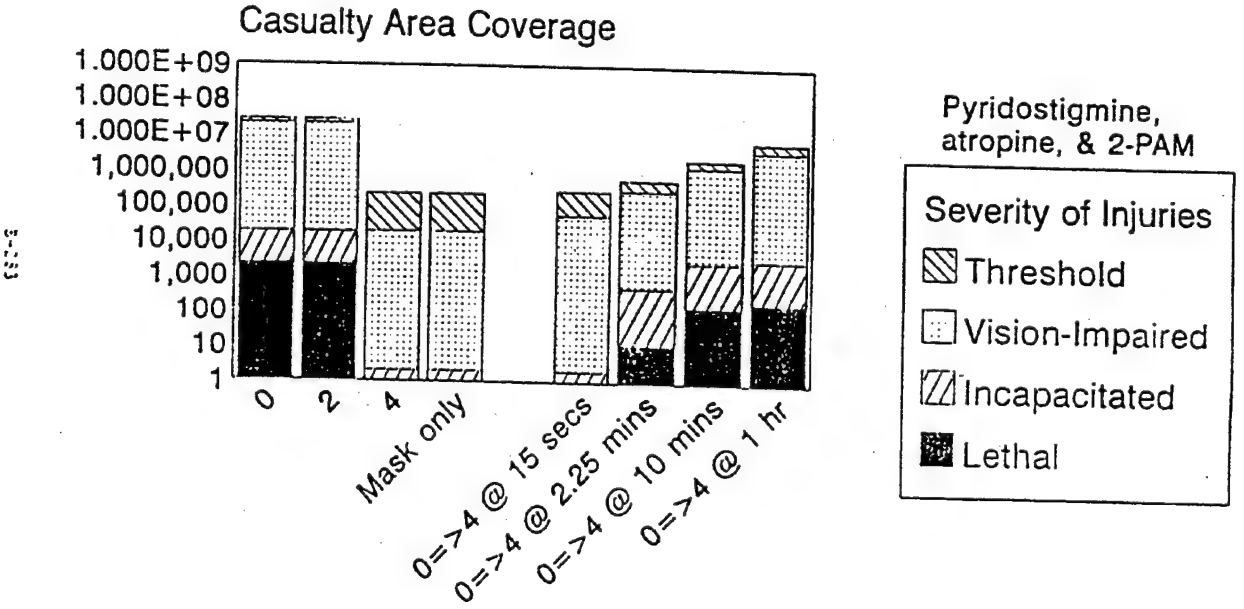


Tactical Ballistic Missile with Submunitions Sarin (GB)



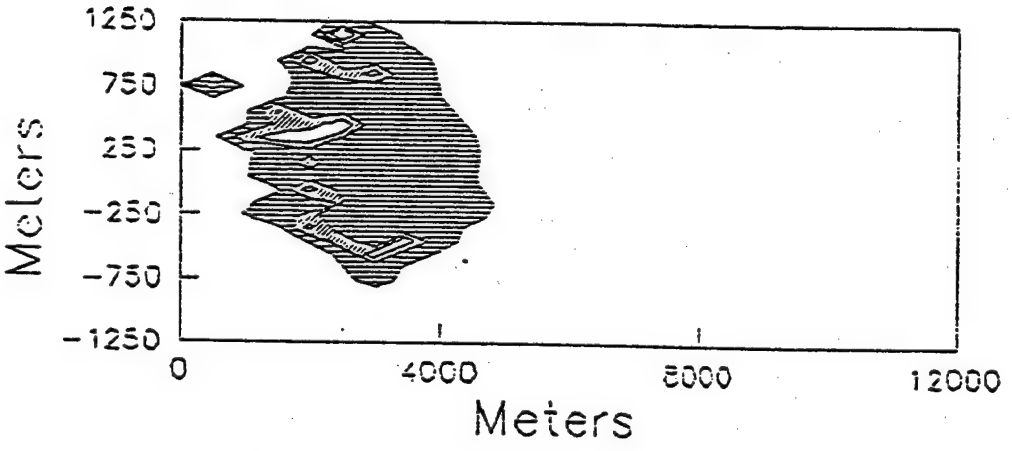
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile with Submunitions Sarin (GB)



4°C (40°F), 1.5m/sec, Stability E

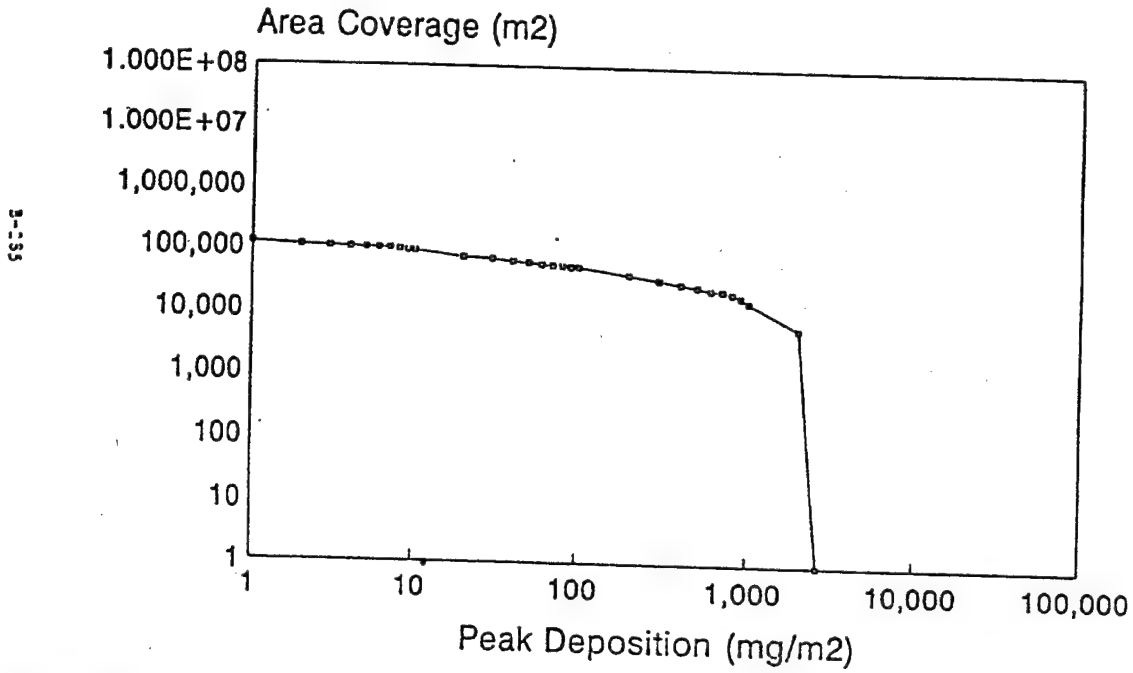
Tactical Ballistic Missile w/ Submunitions
Sarin (GB)



25oC (77oF)
3 m/sec
Stability D

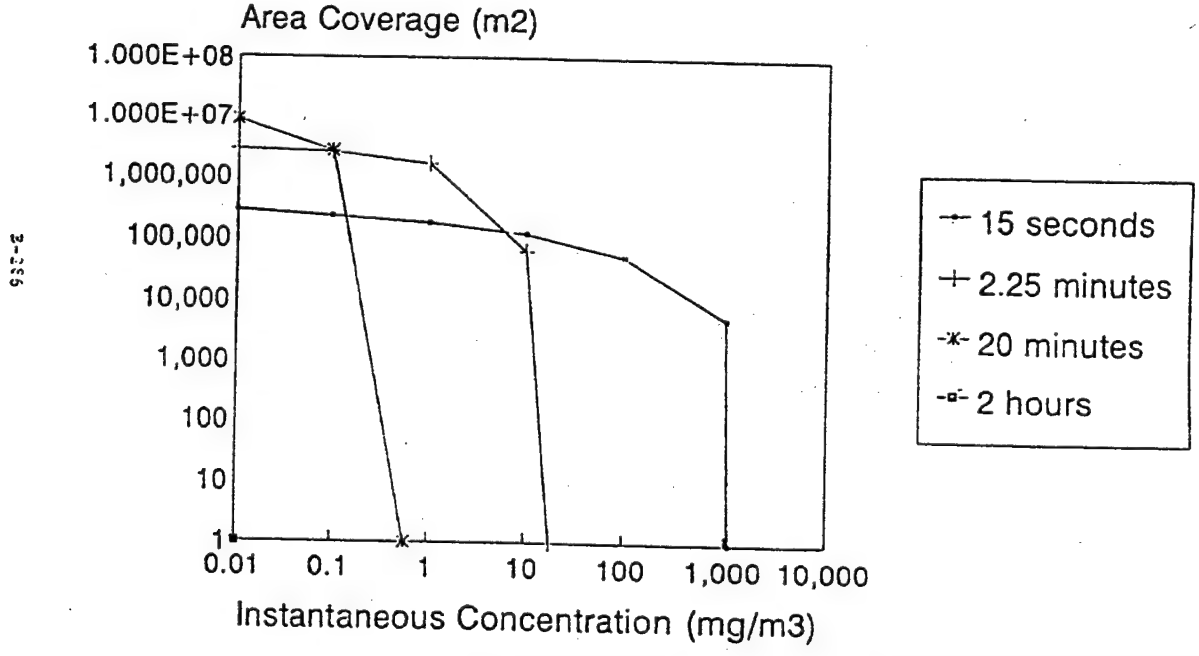
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile w/Submunitions Sarin (GB)



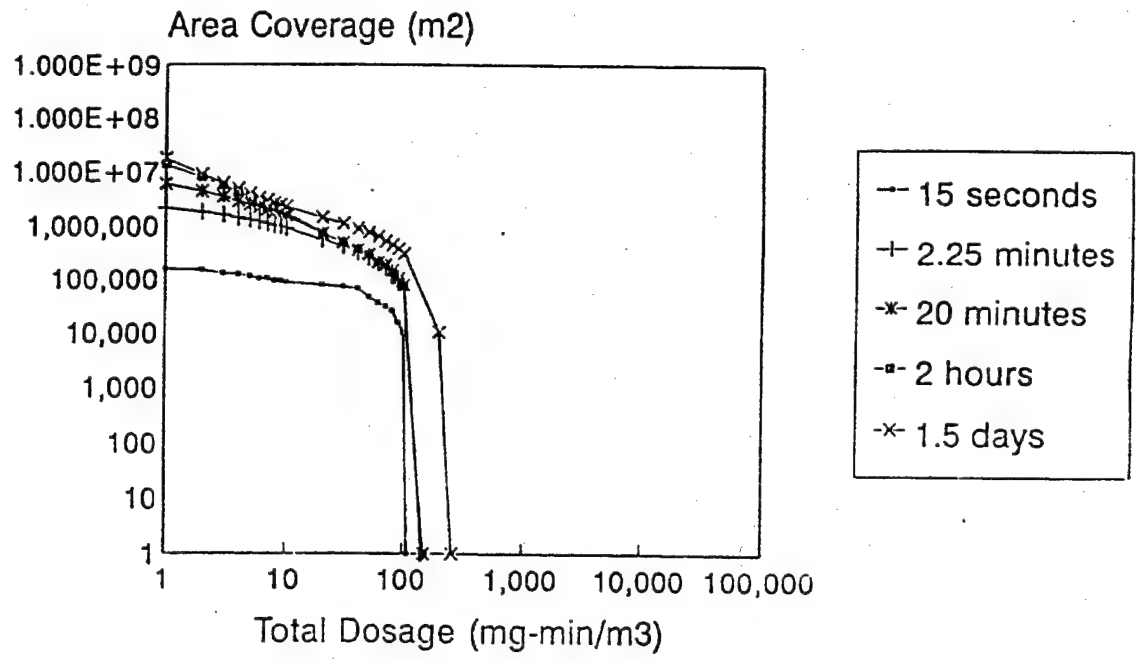
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile w/Submunitions Sarin (GB)



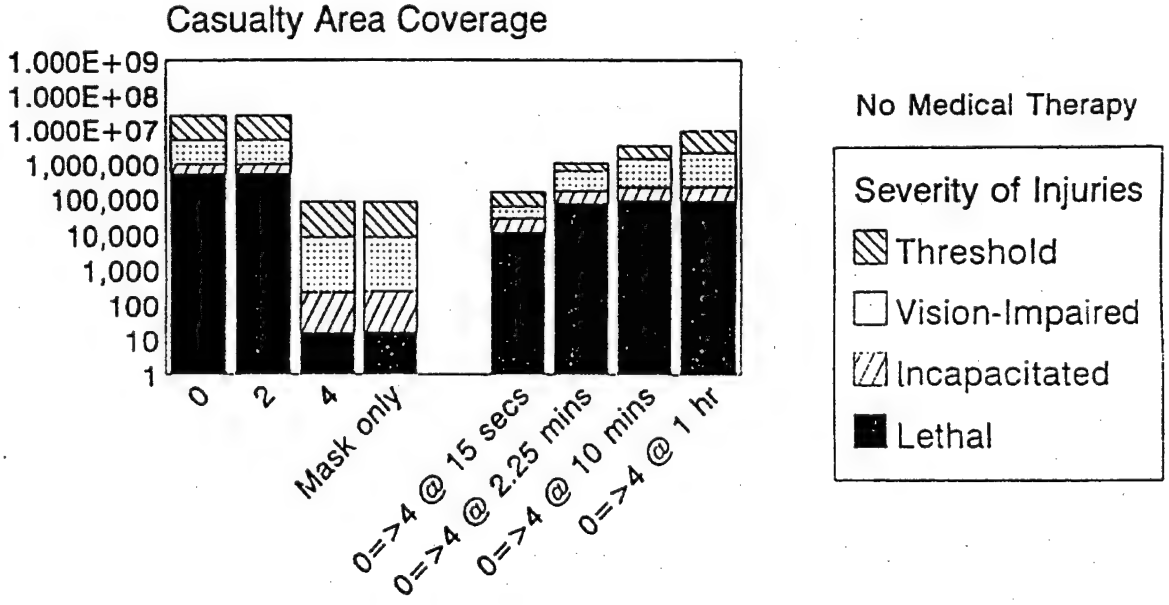
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile w/Submunitions Sarin (GB)



25°C (77°F), 3m/sec, stability D

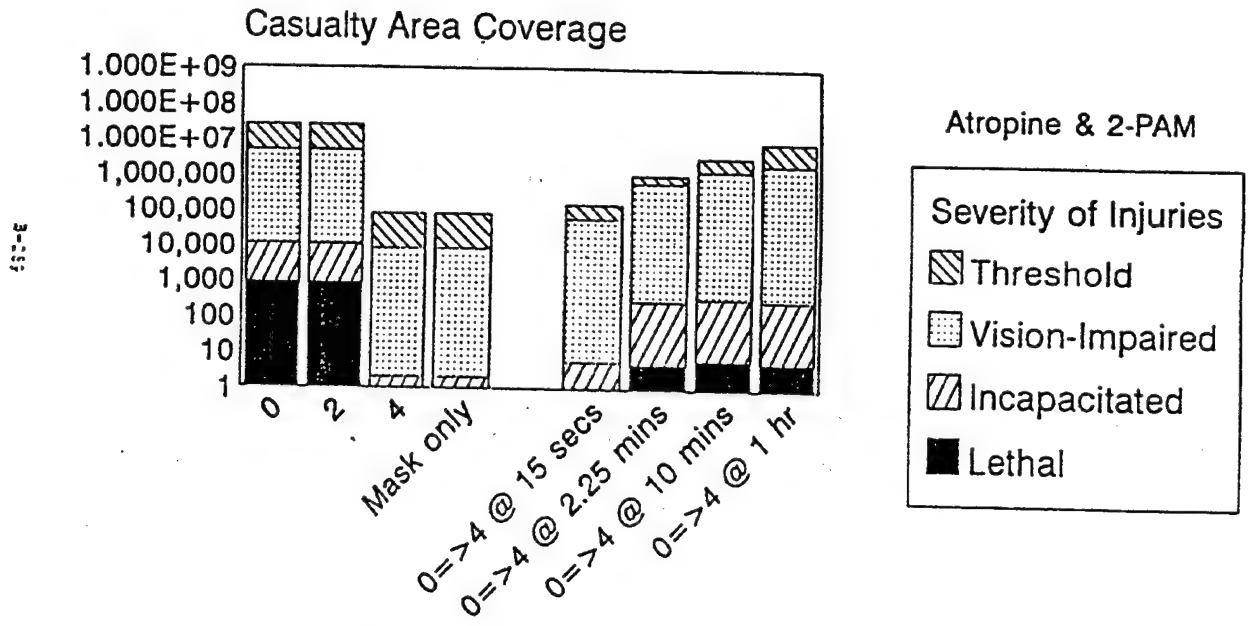
Tactical Ballistic Missile with Submunitions Sarin (GB)



Mission Oriented Protective Posture

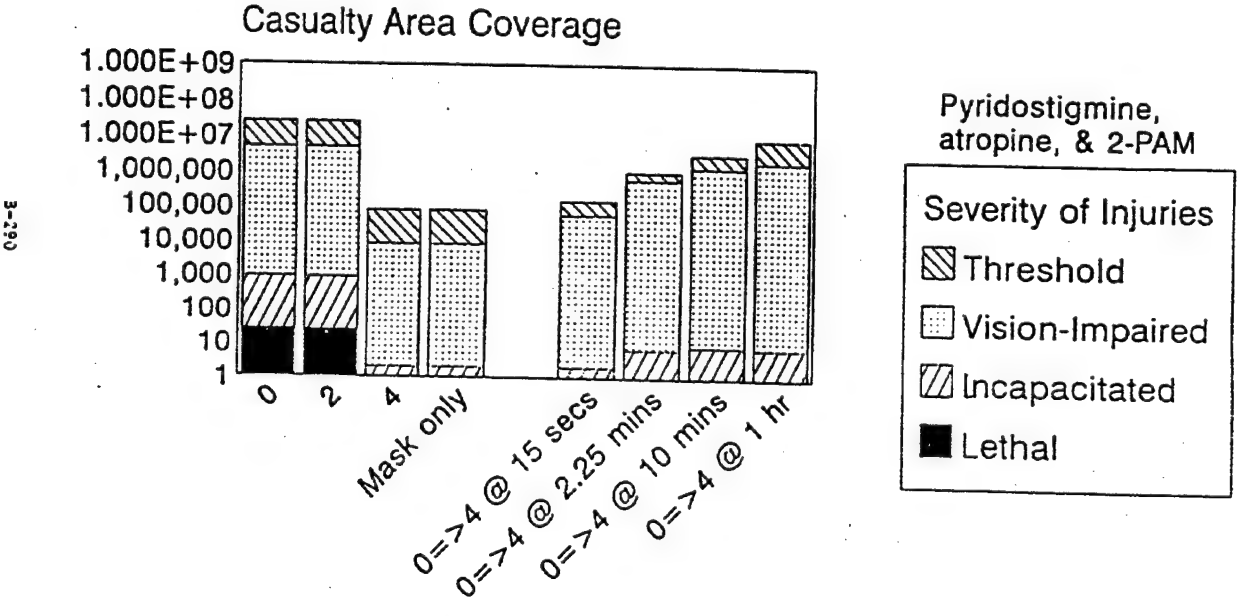
25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile with Submunitions Sarin (GB)



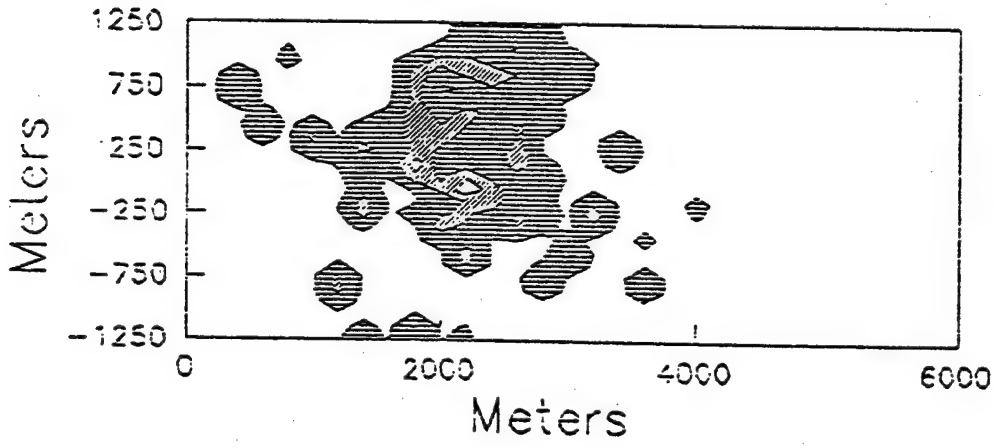
25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile with Submunitions Sarin (GB)



25°C (77°F), 3m/sec, Stability D

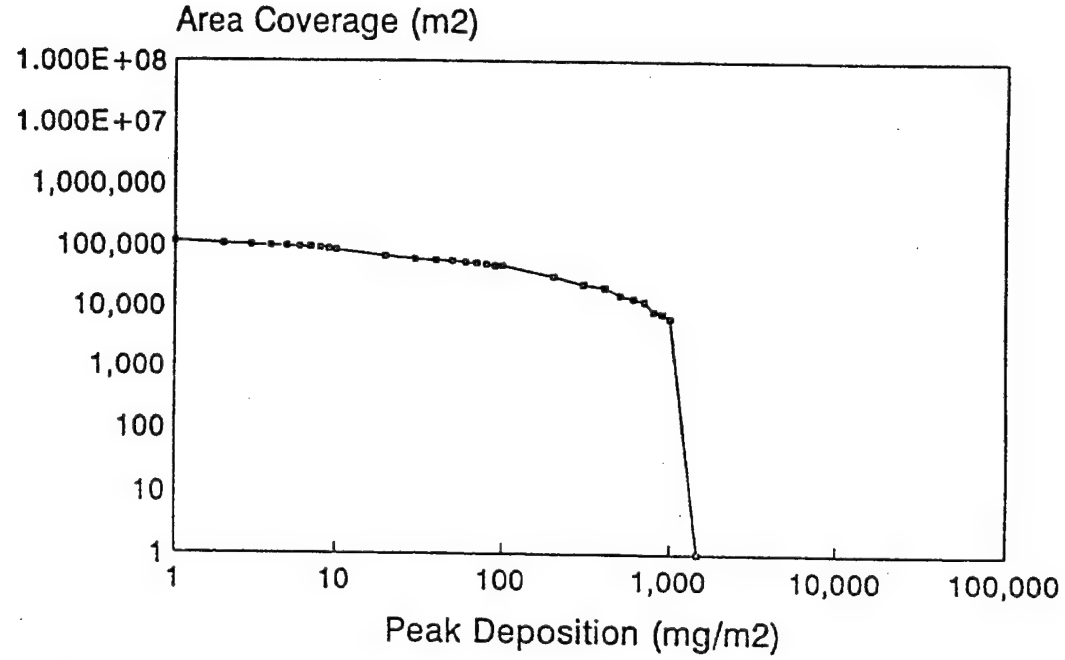
Tactical Ballistic Missile w/ Submunitions
Sarin (GB)



49oC (120oF)
6 m/sec
Stability B

Visually Impaired
Incapacitated
Lethal

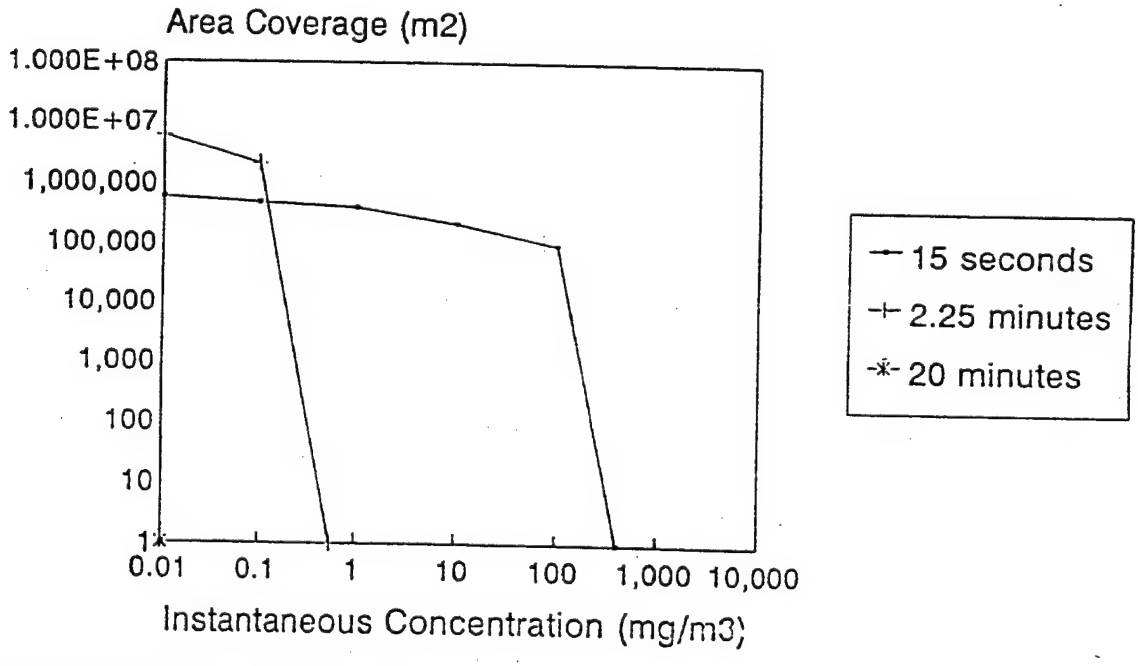
Tactical Ballistic Missile w/Submunitions Sarin (GB)



3-292

49°C (120°F), 6m/sec, stability B

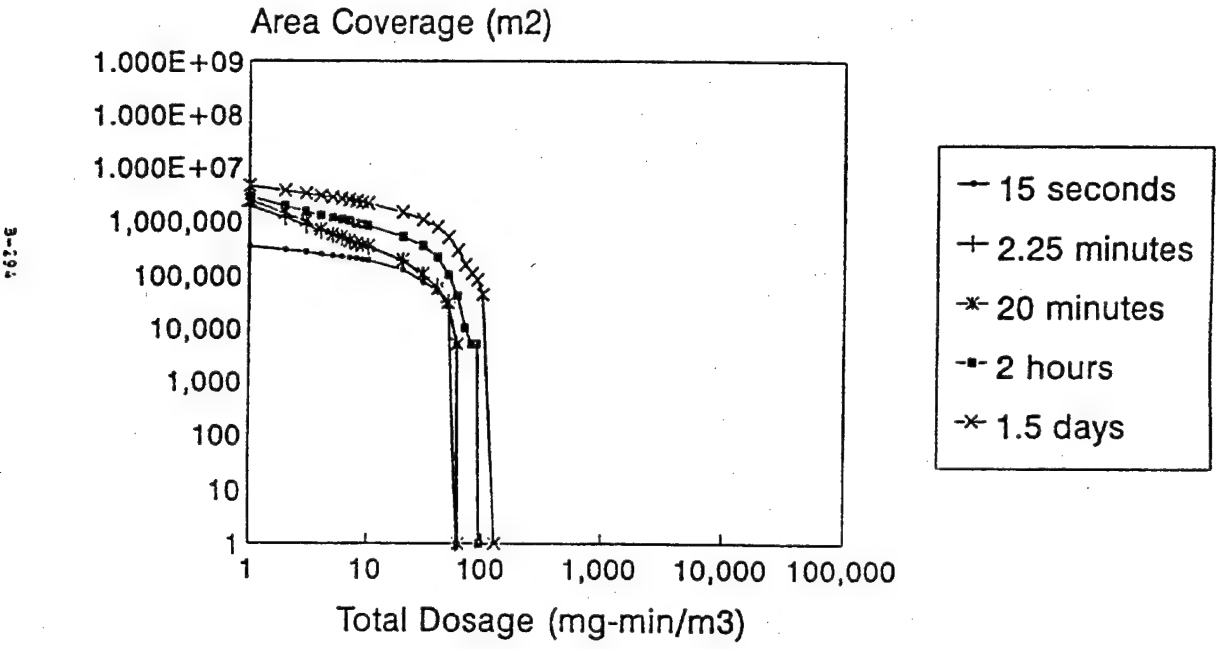
Tactical Ballistic Missile w/Submunitions Sarin (GB)



3-293

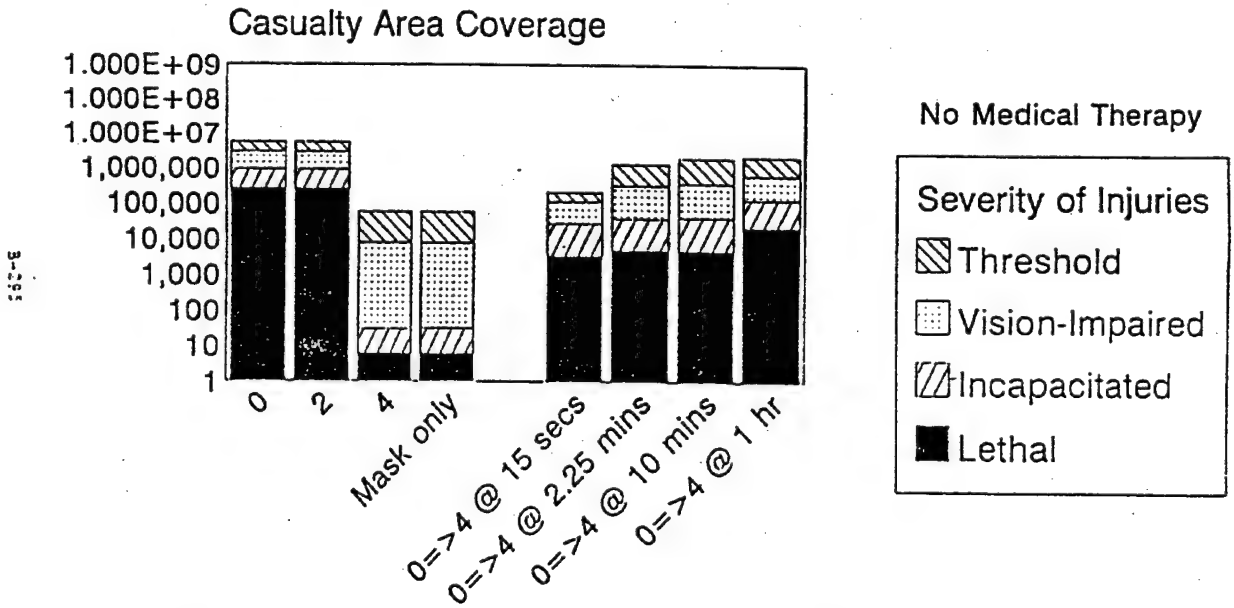
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile w/Submunitions Sarin (GB)



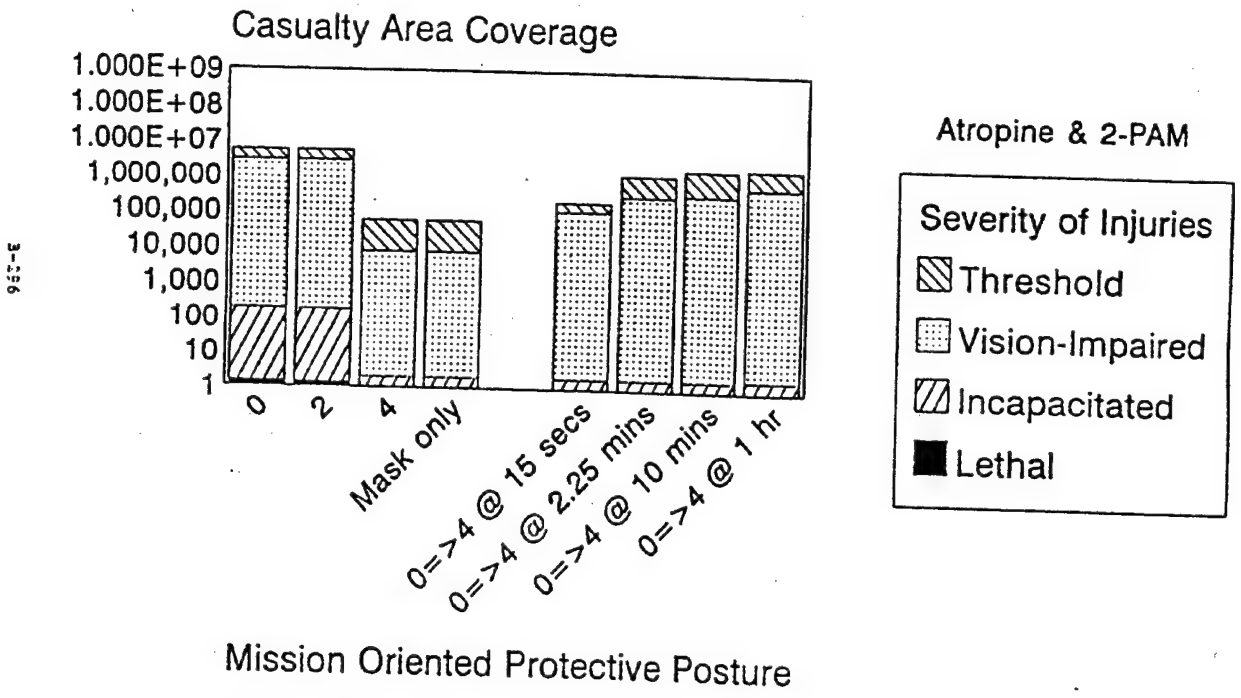
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile with Submunitions Sarin (GB)



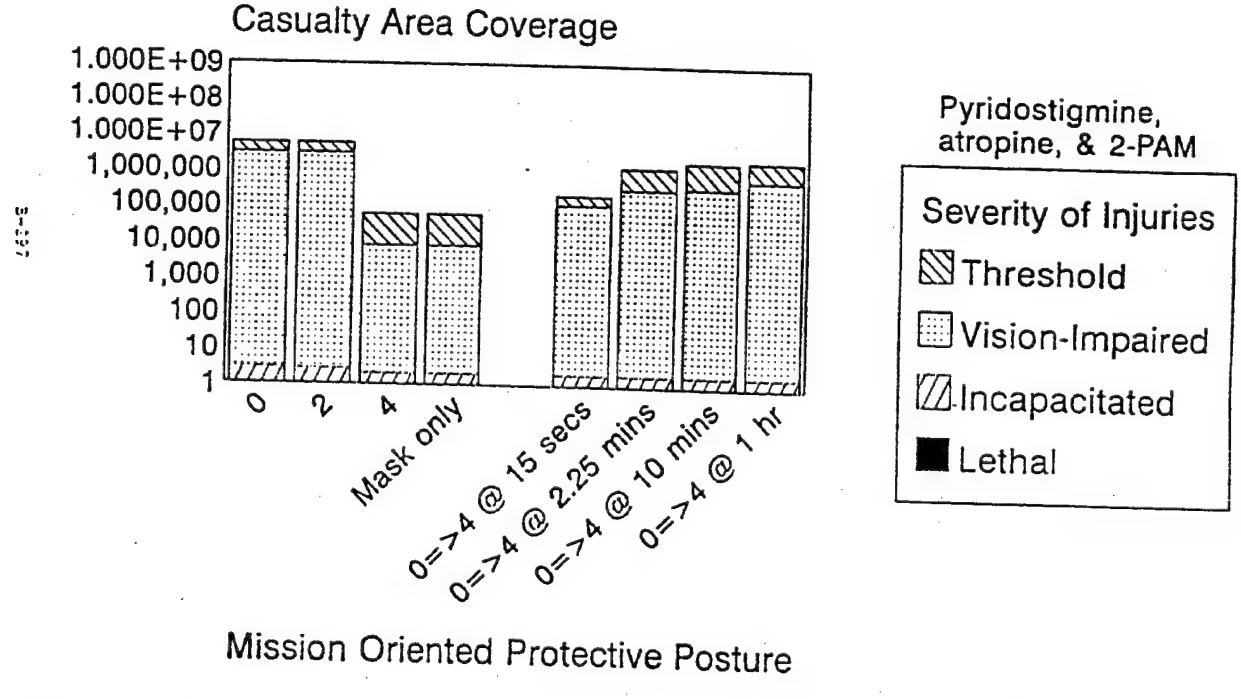
49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
Sarin (GB)



49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
Sarin (GB)



49°C (120°F), 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE WITH SUBMUNITIONS

Soman (GD)

Tactical Ballistic Missile with Submunitions - Soman (GD)

Approximately 100 submunitions, each containing just over 2 kilograms of soman was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The submunitions were released from the tactical ballistic missile at an altitude of approximately 1.5 kilometers producing a 1.2 kilometer diameter submuniton pattern on the ground.

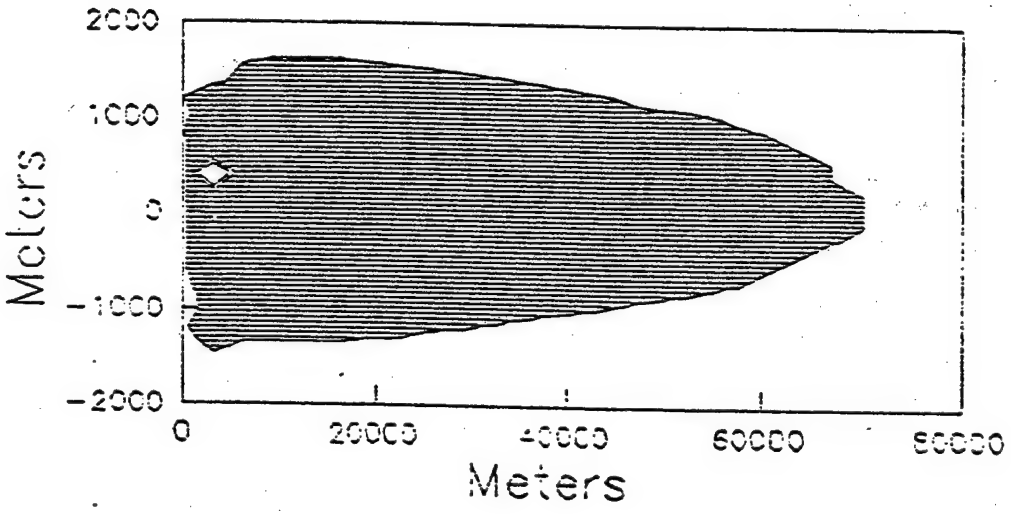
The peak liquid deposition from the attacks was between 1 and 10 grams/square meter with no liquid area coverage just above 0.1 square kilometer under all three of the meteorological cases.

Concentrations dropped below significant levels between 1 hours and less than 16 hours for the low temperature, low windspeed case while and between 3 minutes and 1 hour in both the moderate temperature, moderate windspeed case and the high temperature, high windspeed case.

The peak dosage is between 100 and 1,000 milligram-minutes/cubic meters for all three meteorology conditions. Maximum dosage area coverage is between 10 and 100 square kilometers for the low temperature, low windspeed and the moderate temperature, moderate windspeed cases. The peak dosage is just less than 10 square kilometers for the high temperature case.

Unprotected lethalities for the low temperature and moderate temperature cases were approximately 1 square kilometer while the high temperature cases yielded a likely area coverage of approximately 0.01 square kilometers. There was more than a three order of magnitude reduction (more than a 99.9 per cent reduction) in likely casualty area coverage if the mask is worn. The likely lethal area is very sensitive to the time of masking.

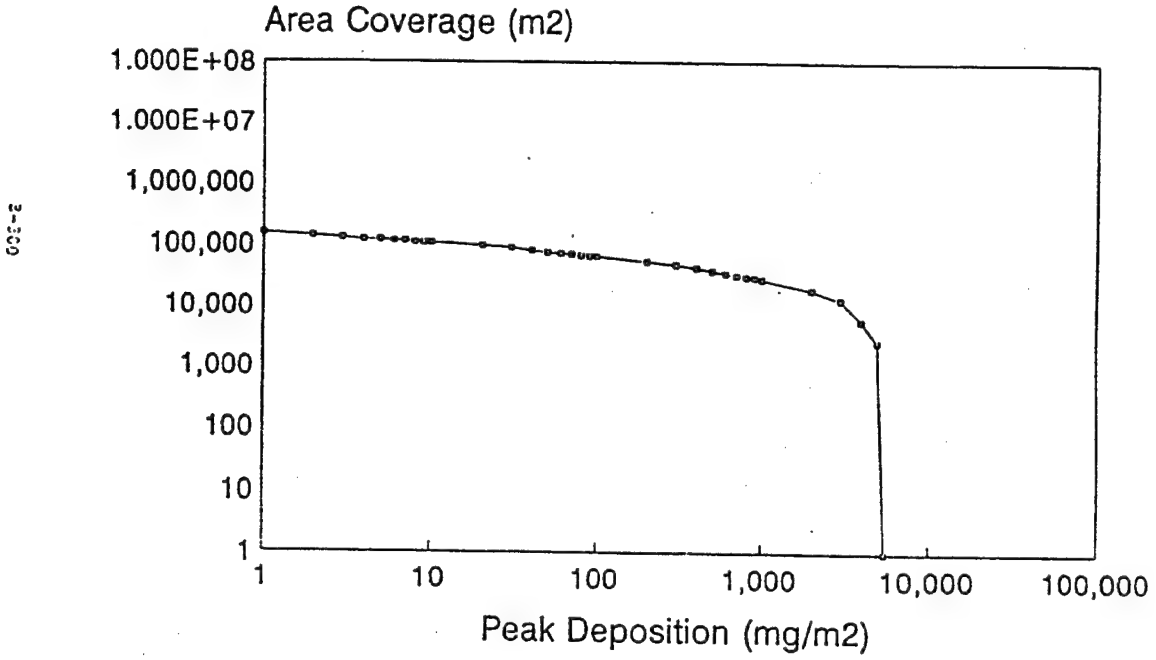
Tactical Ballistic Missile w/Submunitions
Soman (GD)



4oC (40oF)
1.5 m/sec
Stability E

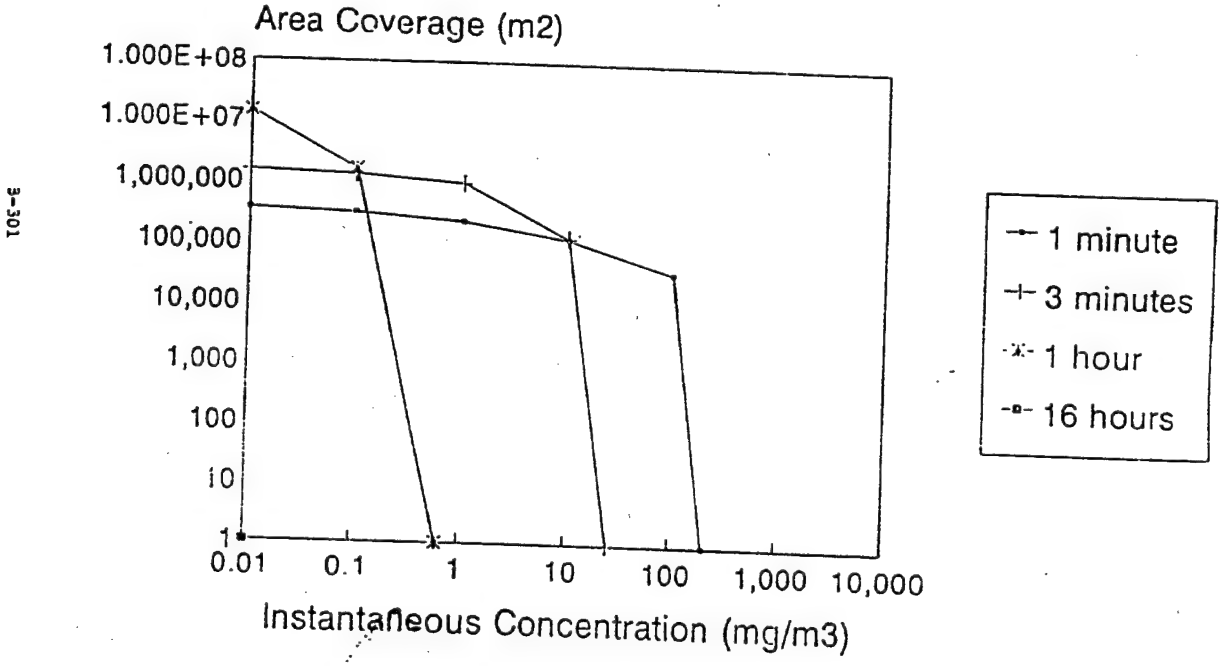
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile w/Submunitions Soman (GD)



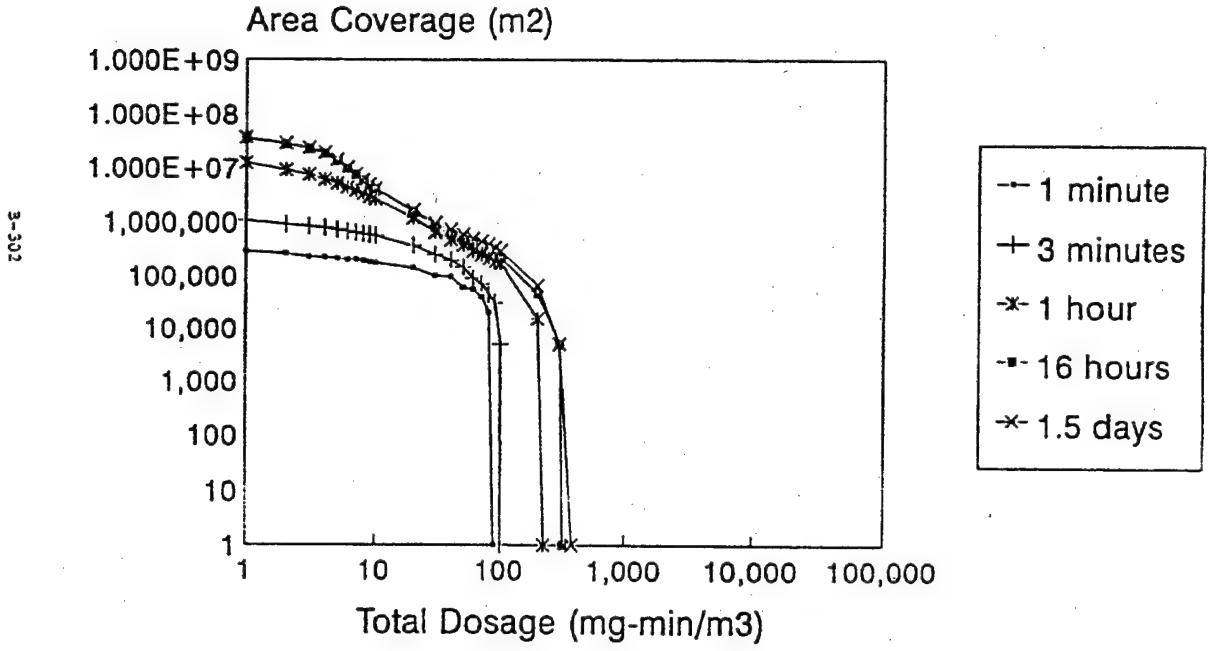
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile w/Submunitions Soman (GD)



4°C (40°F), 1.5m/sec, stability E

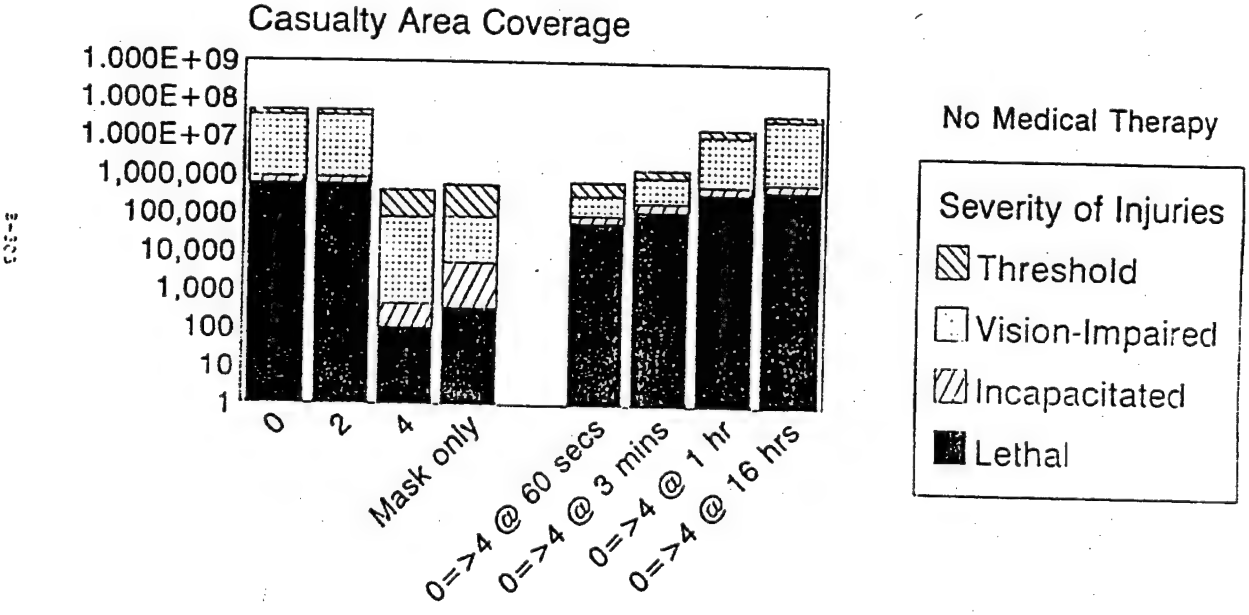
Tactical Ballistic Missile w/Submunitions Soman (GD)



3-302

4°C (40°F), 1.5m/sec, stability E

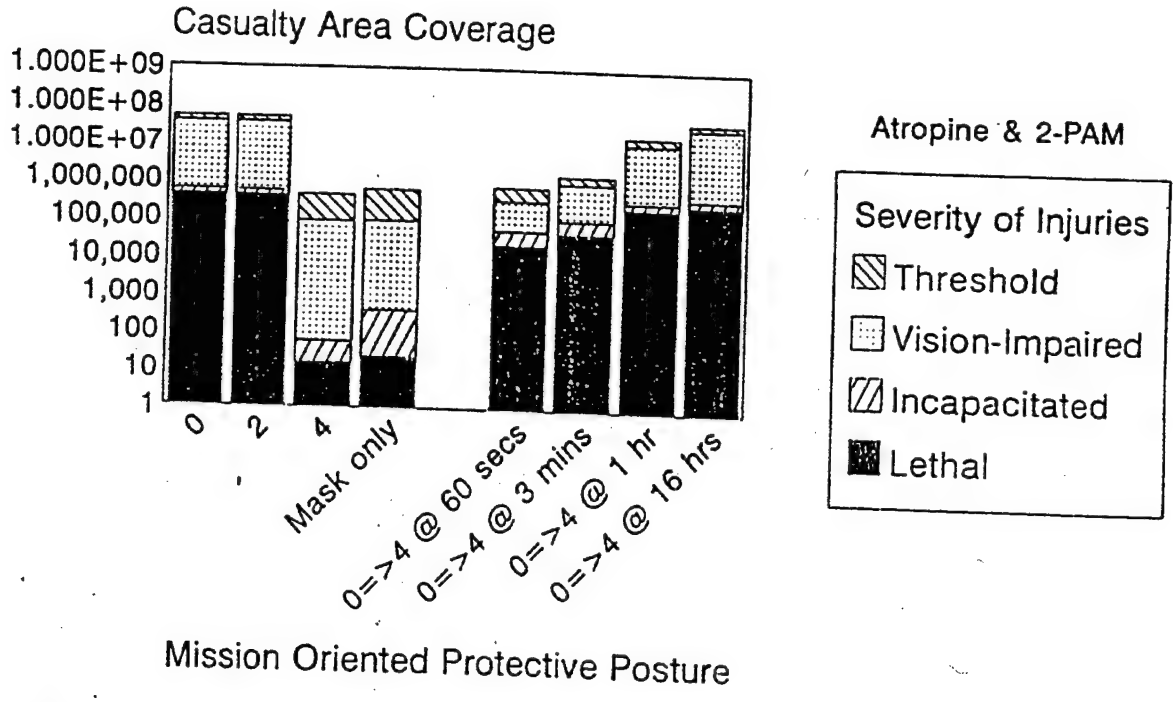
Tactical Ballistic Missile with Submunitions Soman (GD)



3-303

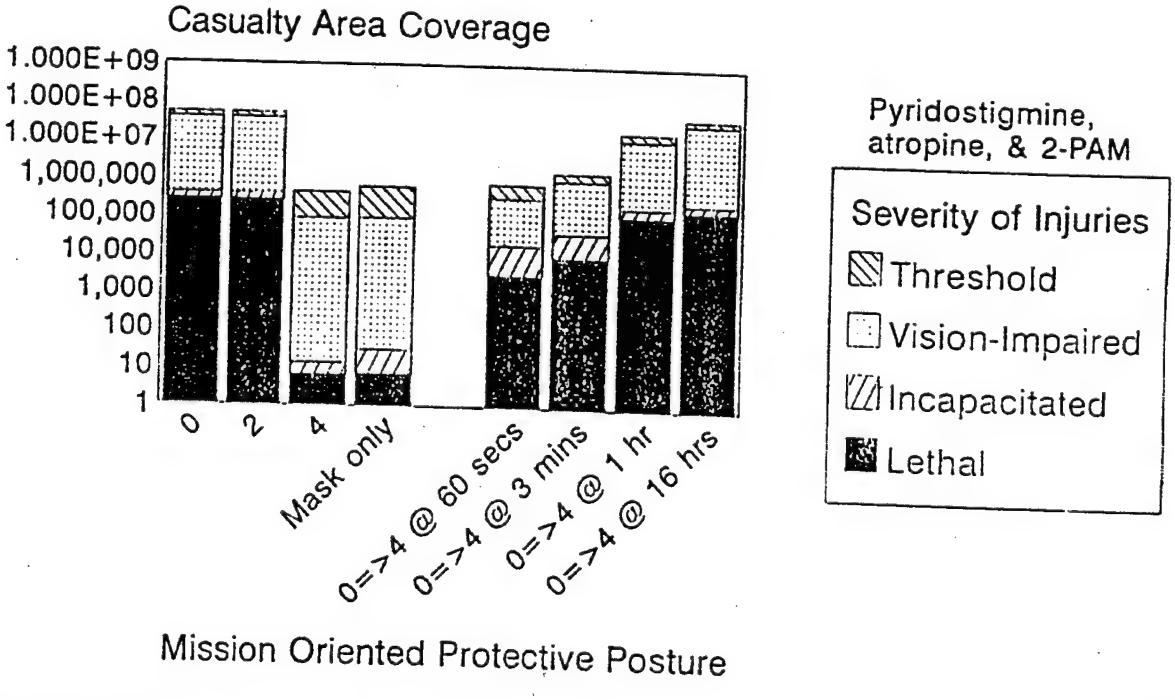
[illegible] Stability E

Tactical Ballistic Missile with Submunitions
Soman (GD)



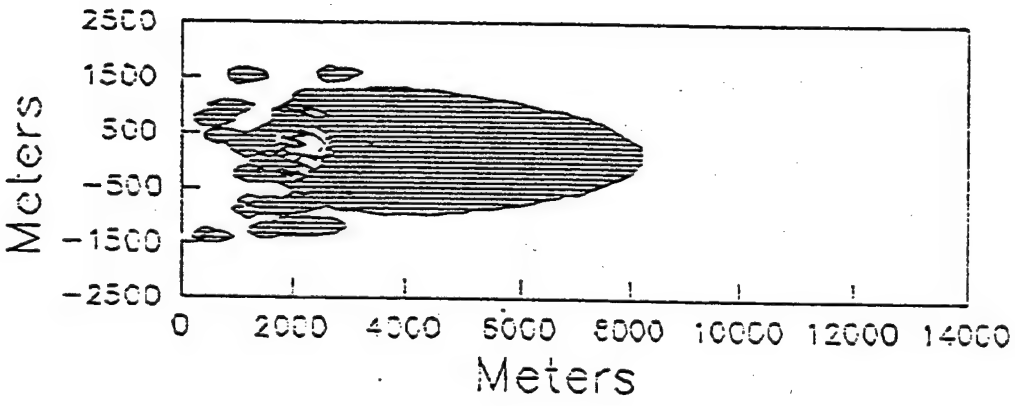
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile with Submunitions
Soman (GD)



4°C (40°F), 1.5m/sec, Stability E

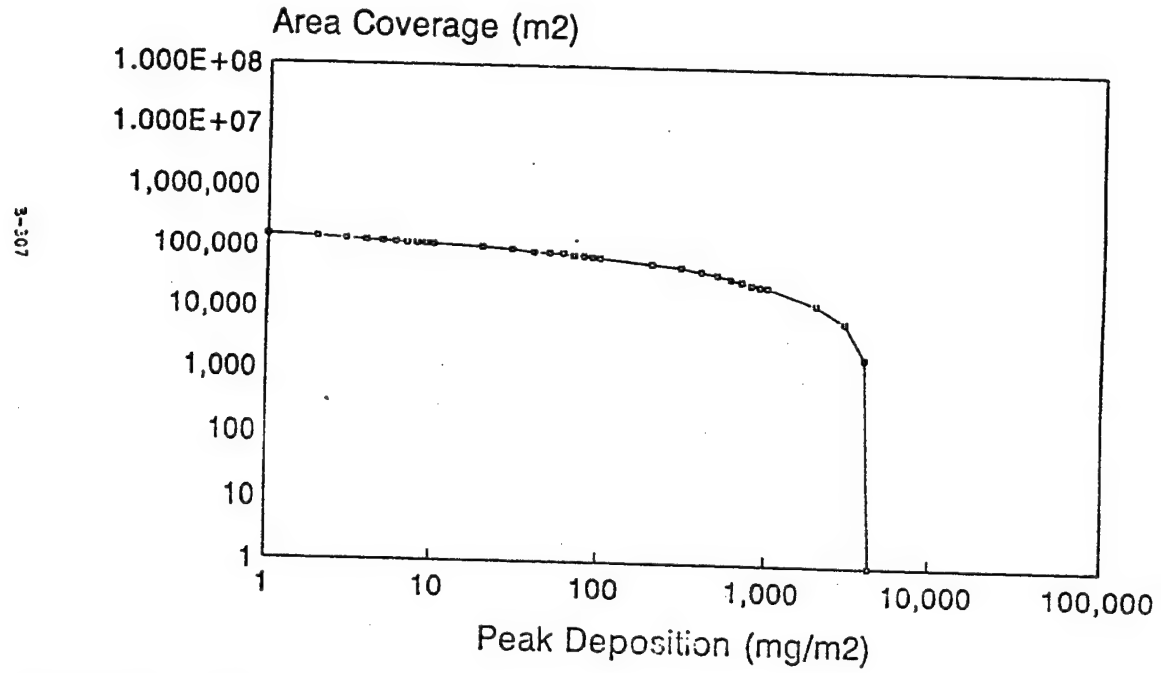
Tactical Ballistic Missile w/Submunitions
Soman (GD)



25oC (77oF)
3 m/sec
Stability D

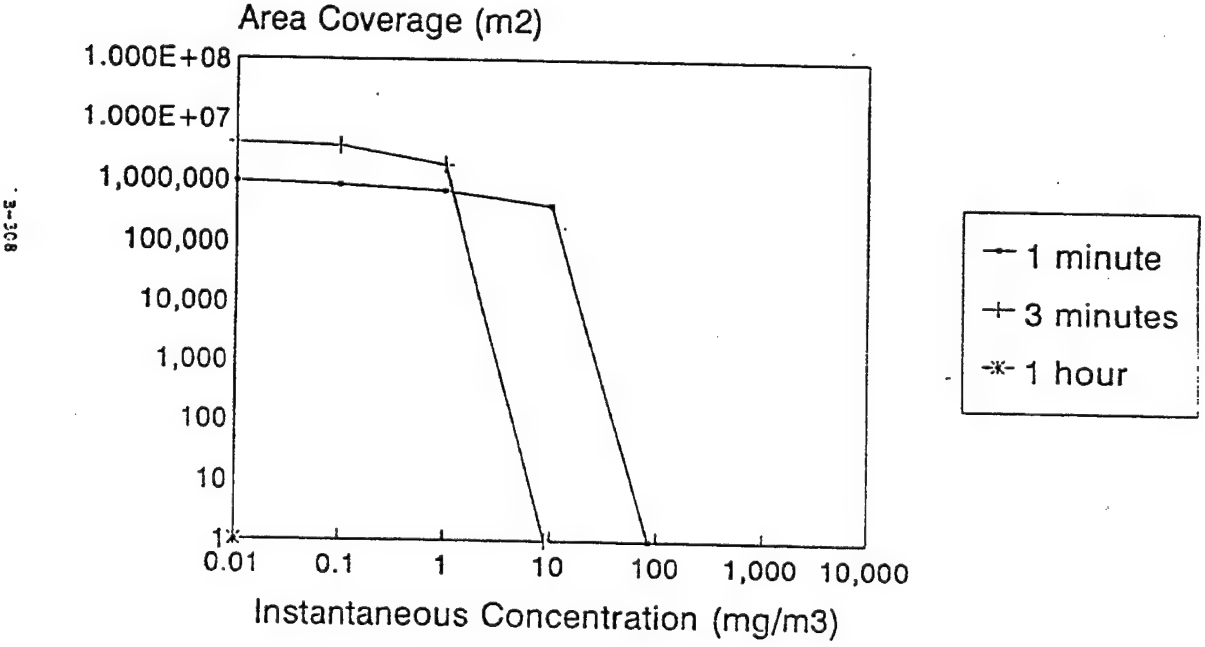
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile w/Submunitions Soman (GD)



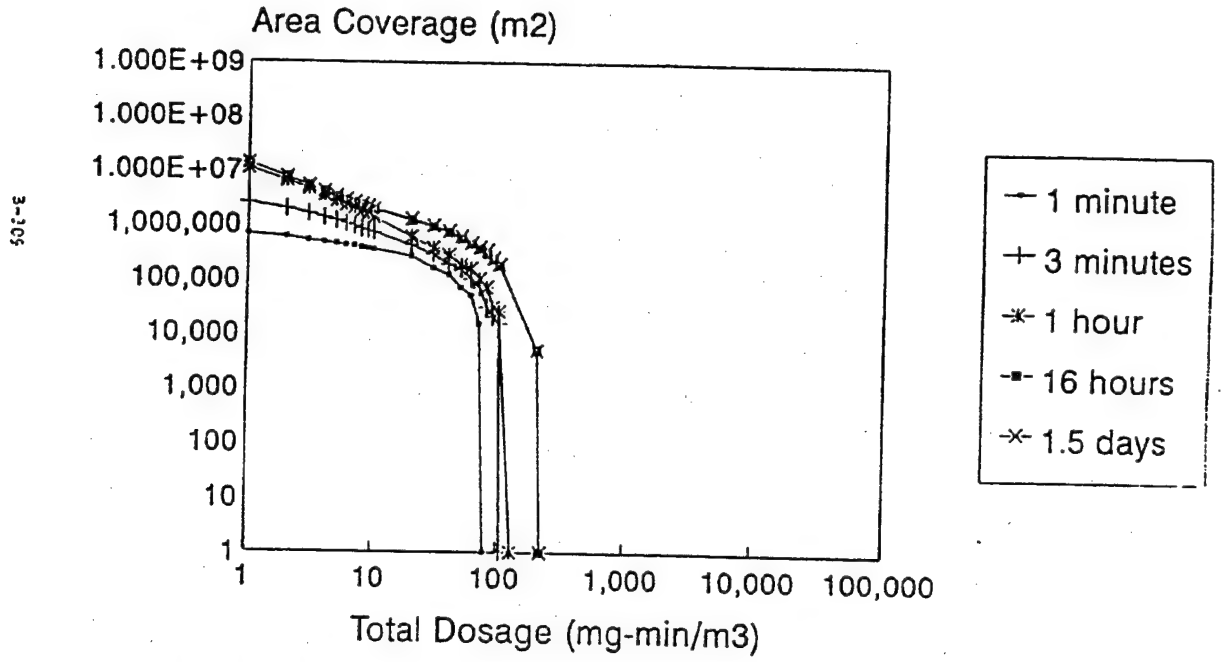
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile w/Submunitions Soman (GD)



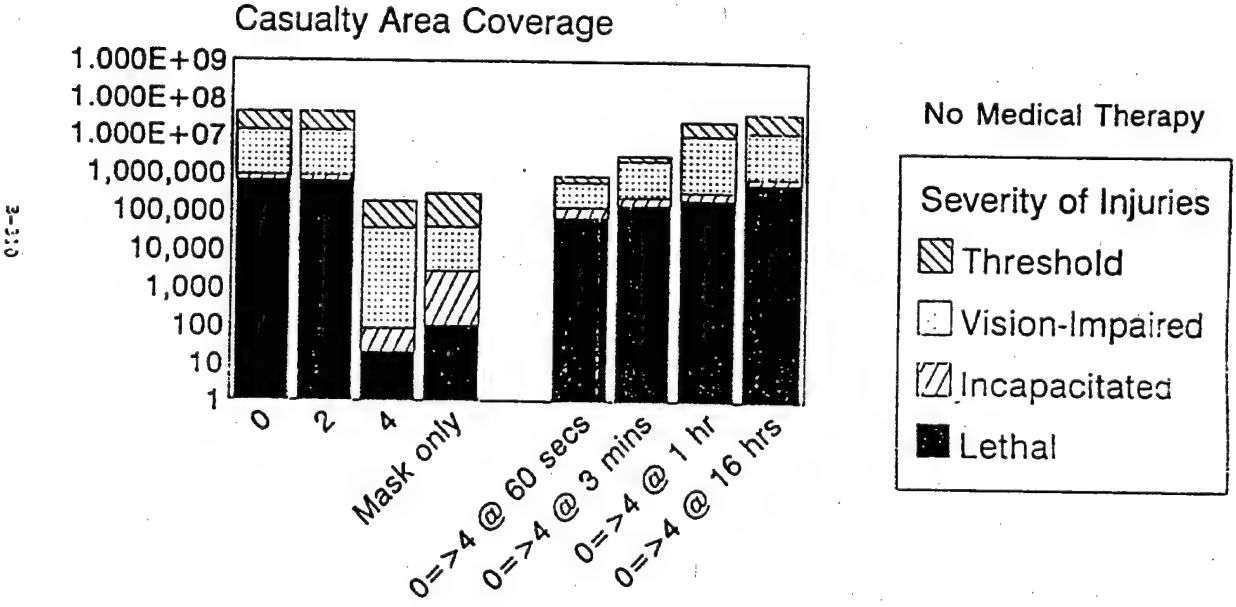
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile w/Submunitions
Soman (GD)



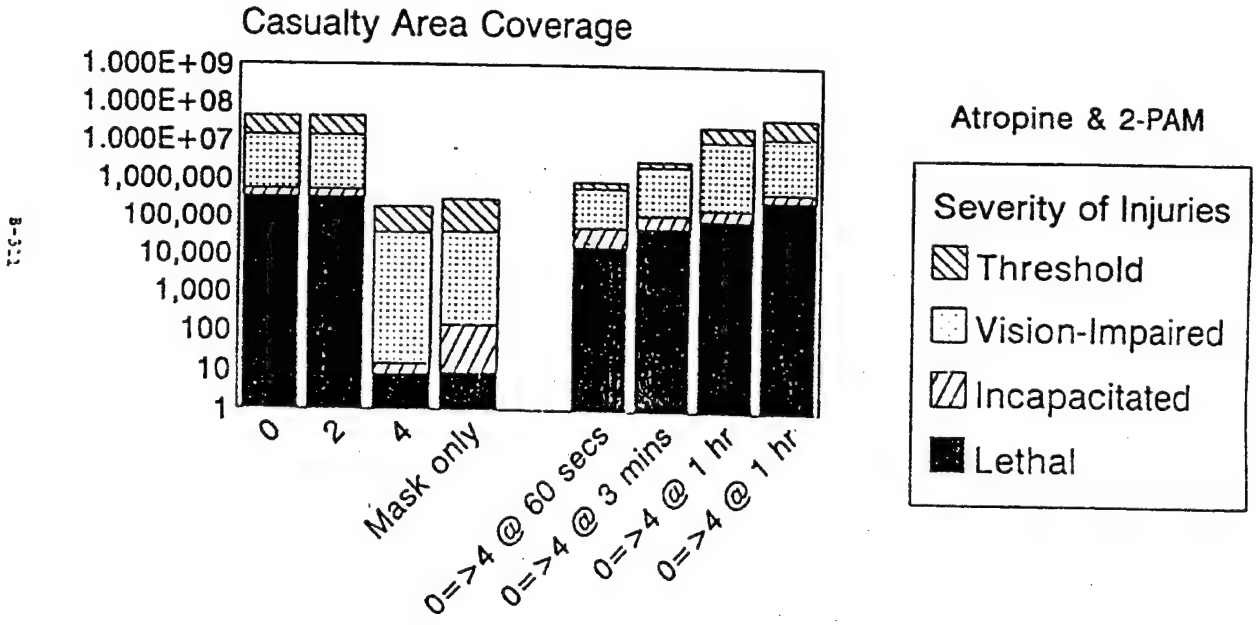
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile with Submunitions
Soman (GD)



25°C (77°F), 3m/sec, Stability D

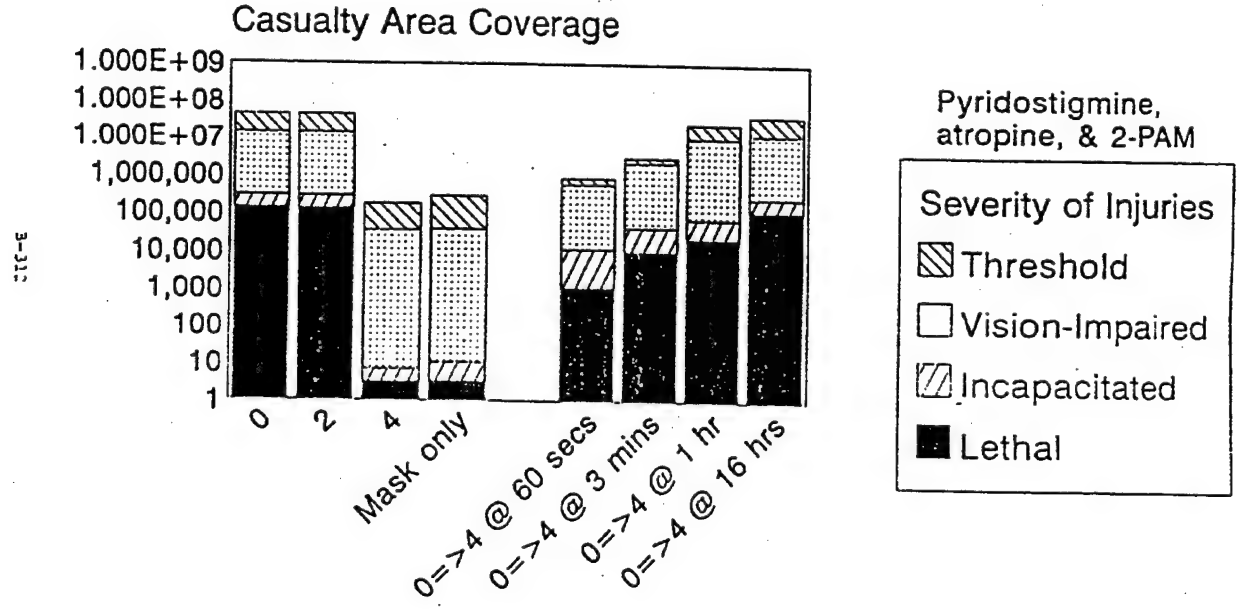
Tactical Ballistic Missile with Submunitions Soman (GD)



Mission Oriented Protective Posture

25°C (77°F), 3m/sec, Stability D

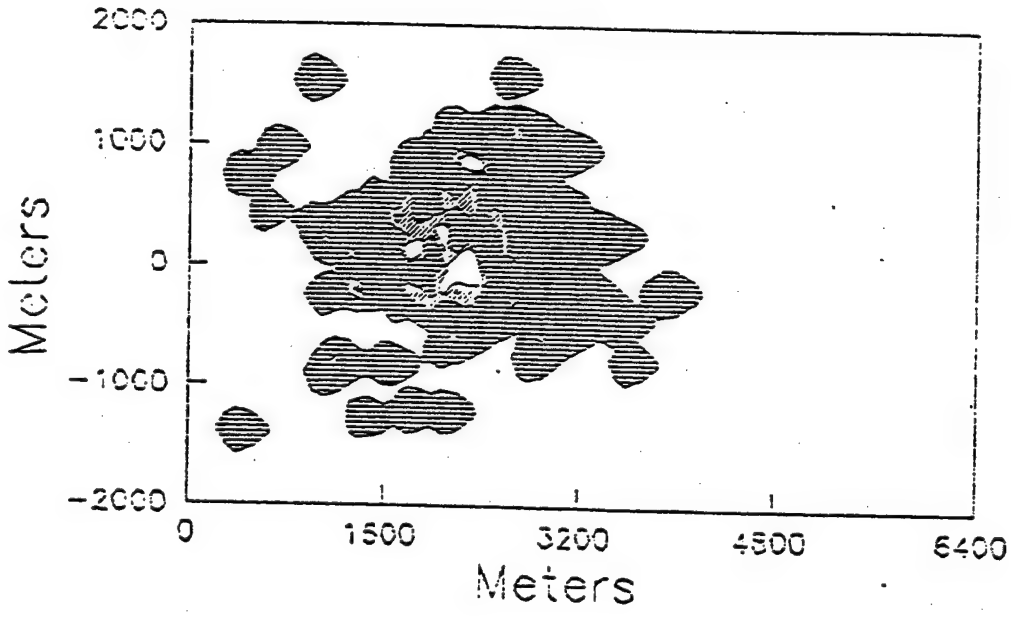
Tactical Ballistic Missile with Submunitions Soman (GD)



Mission Oriented Protective Posture

25°C (77°F), 3m/sec, Stability D

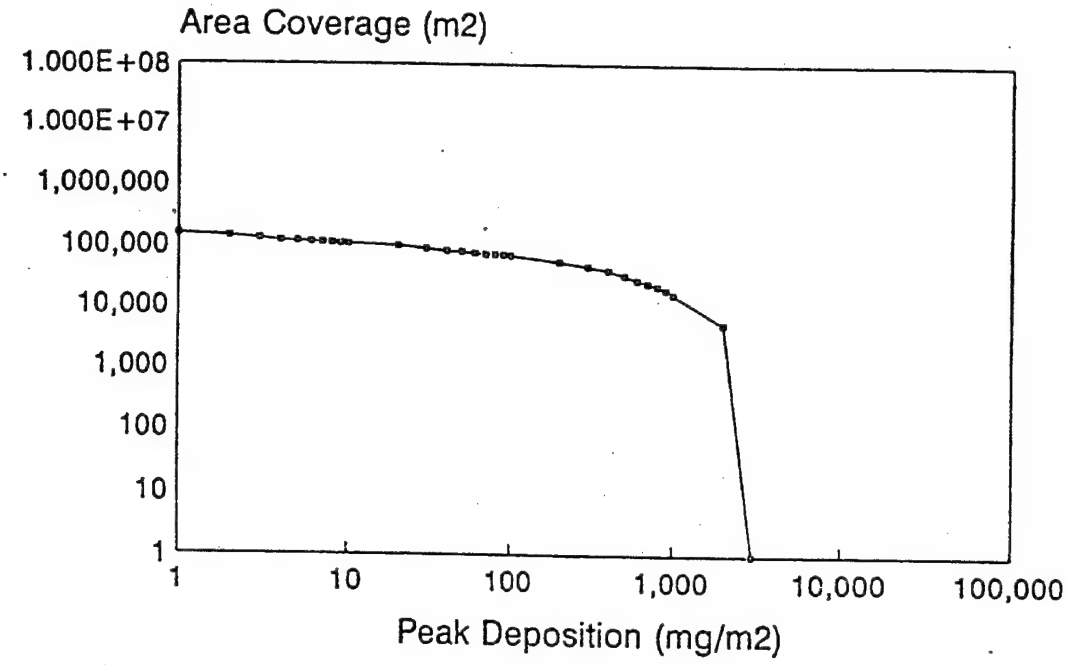
Tactical Ballistic Missile w/ Submunitions
Soman (GD)



49oC (120oF)
6 m/sec
Stability B

Visually Impaired
Incapacitated
Lethal

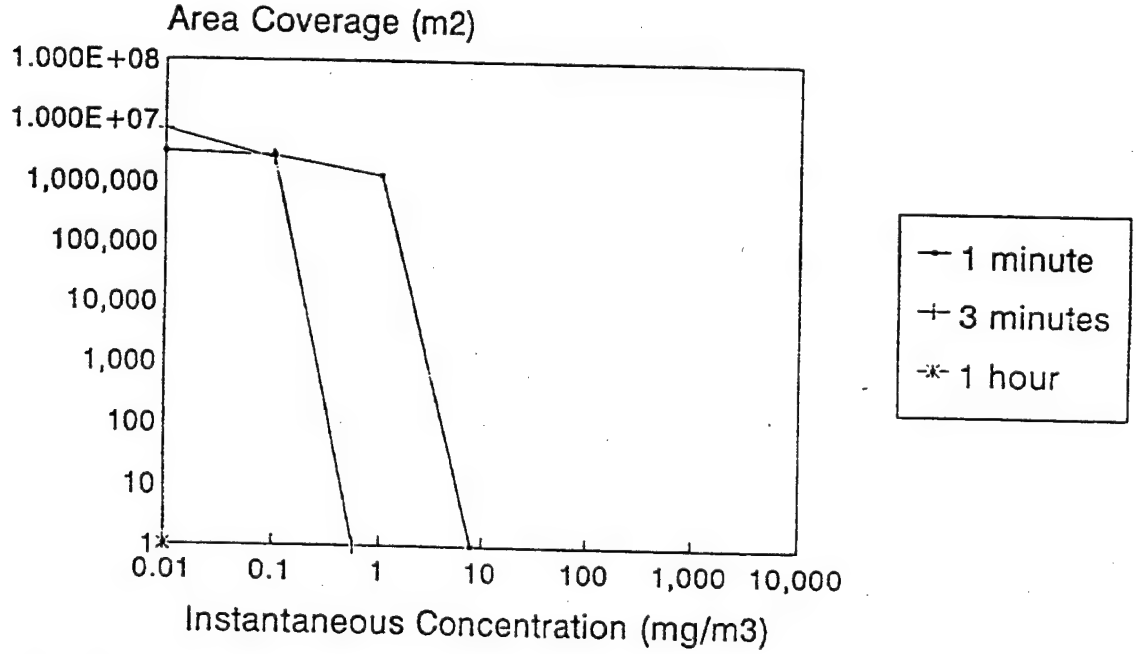
Tactical Ballistic Missile w/Submunitions Soman (GD)



B-314

49°C (120°F), 6m/sec, stability B

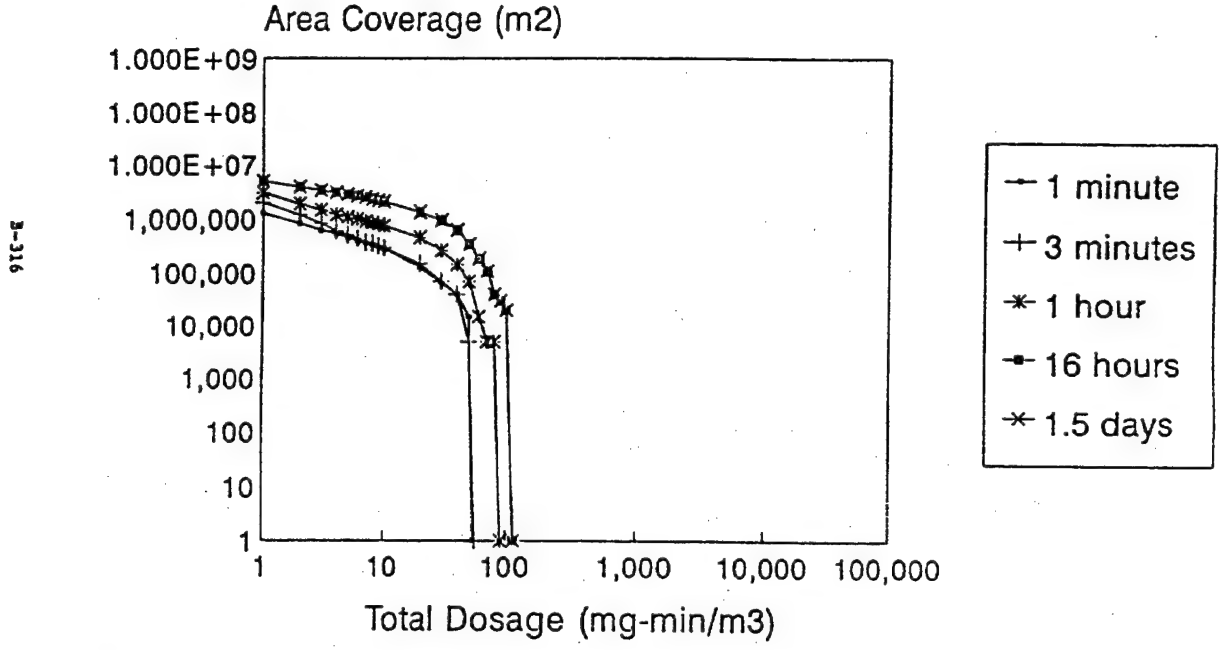
Tactical Ballistic Missile w/Submunitions Soman (GD)



B-315

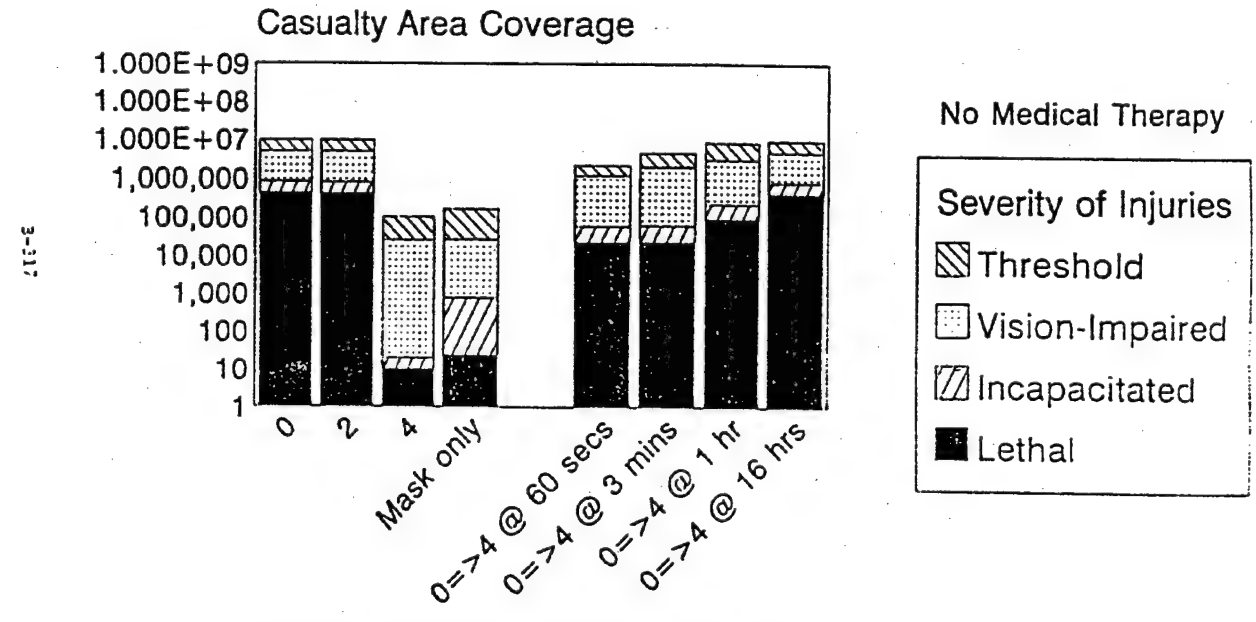
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile w/Submunitions Soman (GD)



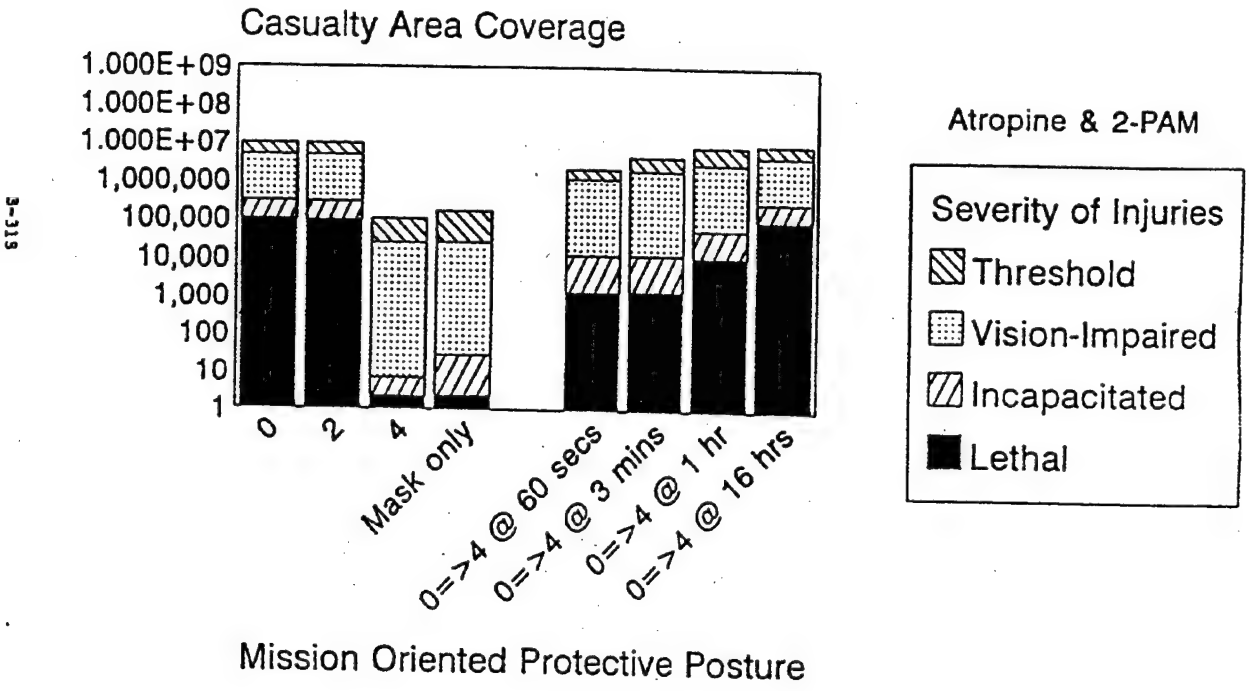
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile with Submunitions Soman (GD)



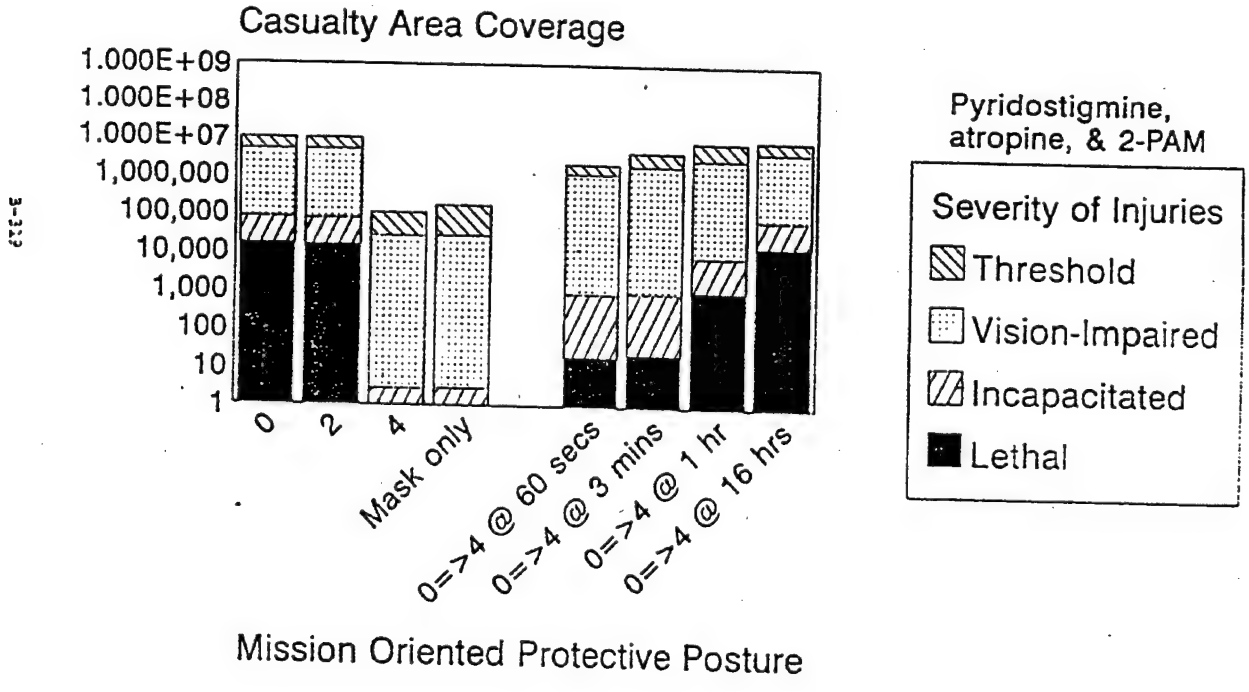
49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
Soman (GD)



49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
Soman (GD)



49°C (120°F), 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE WITH SUBMUNITIONS

VX

Tactical Ballistic Missile with Submunitions - VX

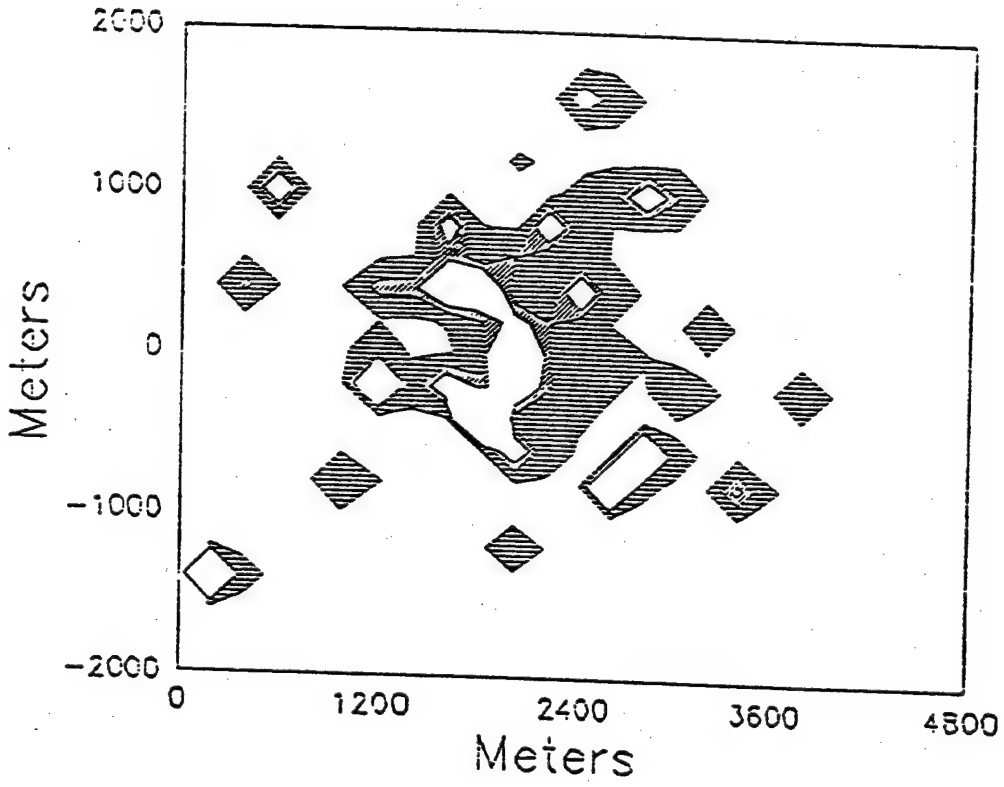
Approximately 100 submunitions, each containing just over 2 kilograms of VX was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The submunitions were released from the tactical ballistic missile at an altitude of approximately 1.5 kilometers producing a 1.2 kilometer diameter submunition pattern on the ground.

The peak liquid deposition from the attacks approached 1 to 2 grams/square meter with no liquid area coverage greater than 10 square kilometer under any of the three meteorological cases.

The volatility of VX is so low that there is little vapor produced in the period of weeks to months of agent evaporation; therefore no agent concentration or dosage area coverage charts are necessary for this attack.

The casualty area coverage charts show the casualty potential of the liquid dissemination. The significant area coverage occurs over approximately 1 square kilometer in all three of the meteorological cases in MOPP 0, MOPP 2, and Mask only clothing configurations. In the case of no medical therapy, most of these casualties would be lethal. Appropriate use of Atropine & 2-PAM produces approximately a two order of magnitude reduction (or a 99 per cent reduction) in likely lethal area coverage without an overgarment and three orders of magnitude reduction (or a 99.9 per cent reduction) in likely lethal area coverage when the overgarment is worn as in MOPP 2. Use of the pyridostigmine pretreatment reduces the likely lethal areas by three orders of magnitude (or a 99.9 per cent reduction) for MOPP 0 cases and by four orders of magnitude (or a 99.99 per cent reduction) for MOPP 2 cases.

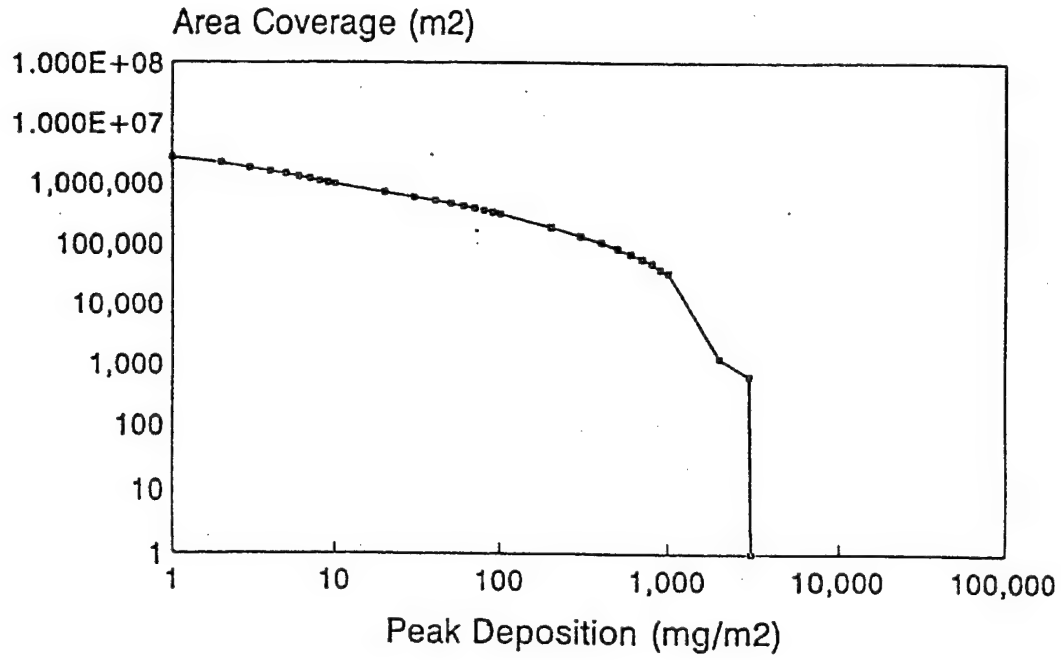
Tactical Ballistic Missile w/ Submunitions
VX



4oC (40oF)
1.5 m/sec
Stability E

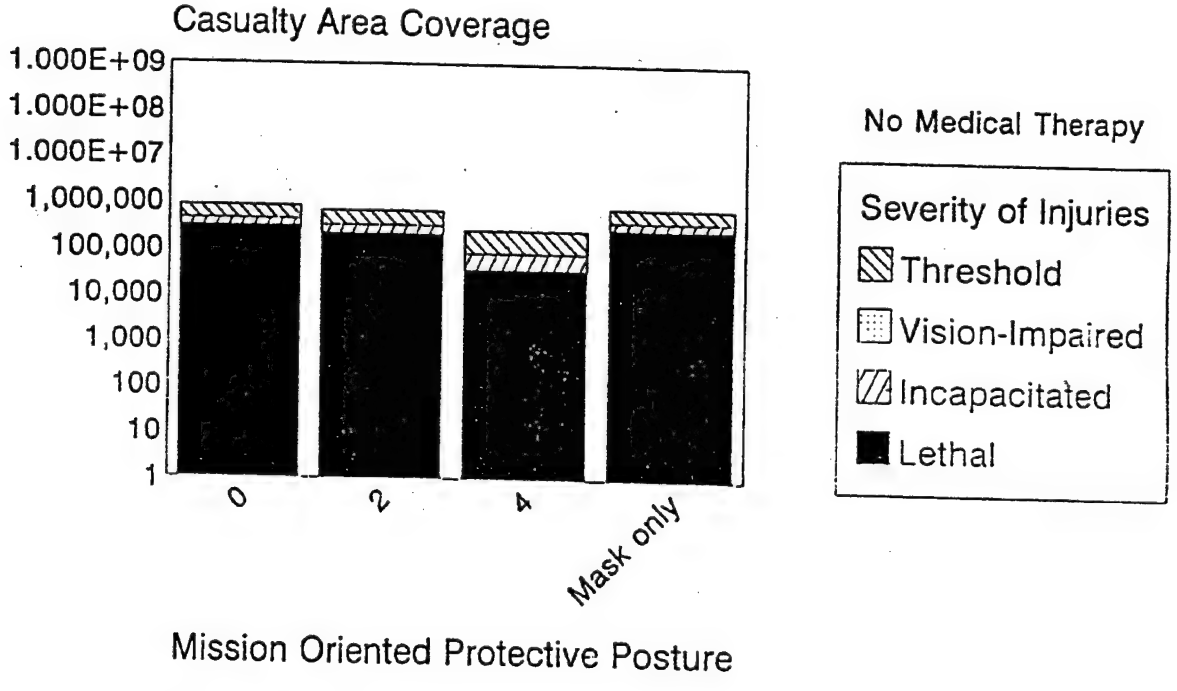
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile w/Submunitions
VX



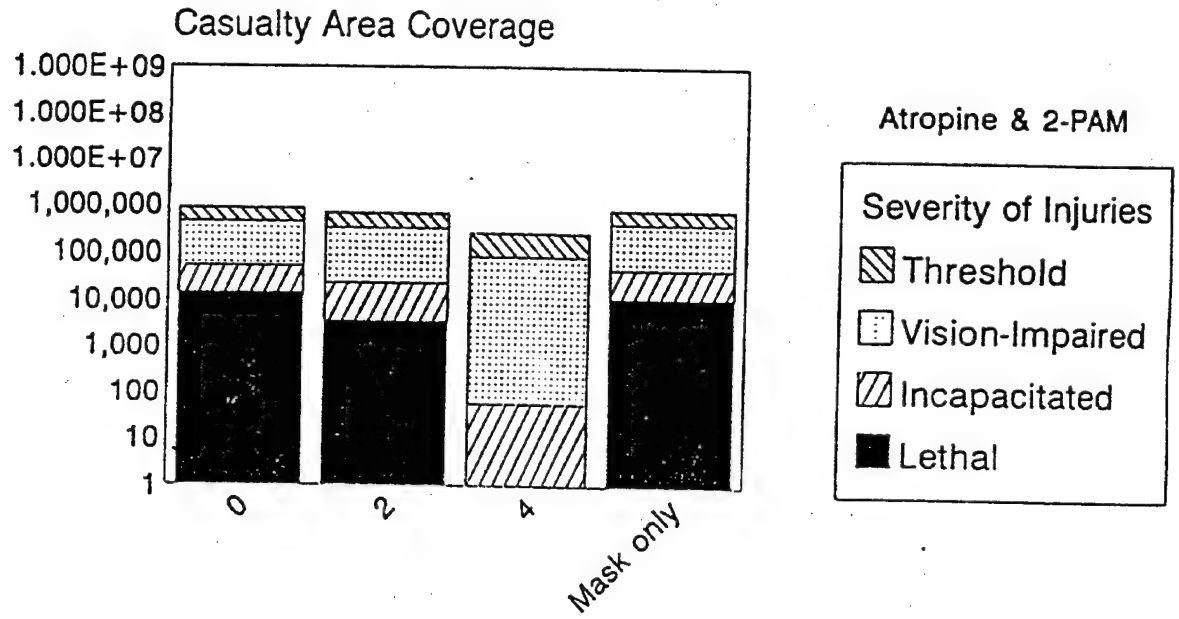
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile with Submunitions
VX



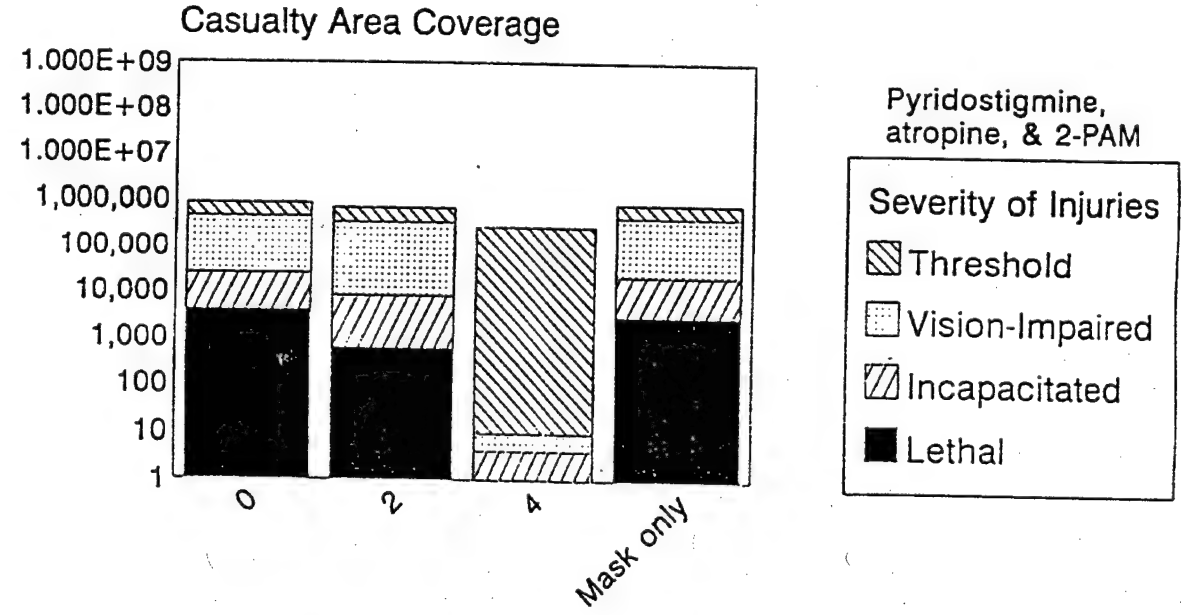
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile with Submunitions
VX



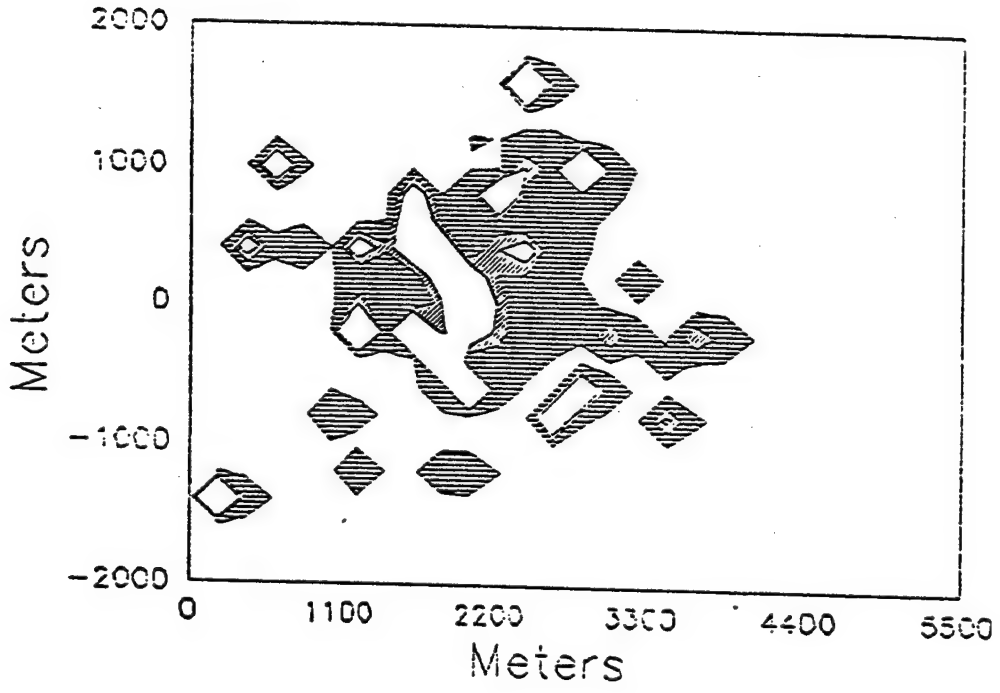
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile with Submunitions
VX



4°C (40°F), 1.5m/sec, Stability E

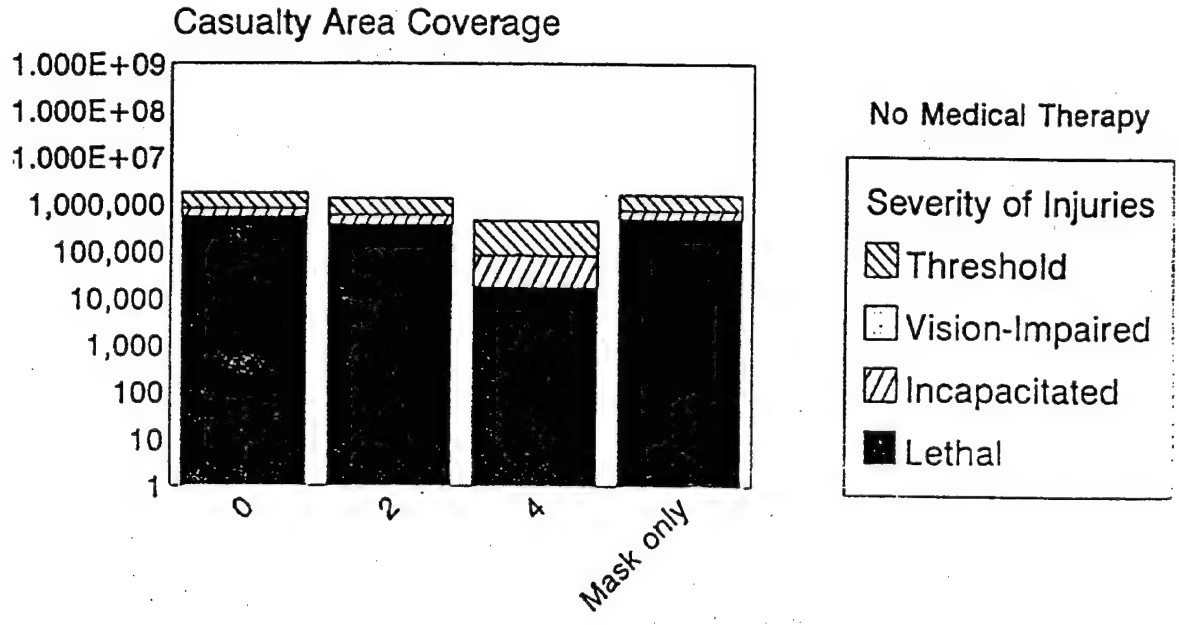
Tactical Ballistic Missile w/ Submunitions
VX



25oC (77oF)
3 m/sec
Stability D

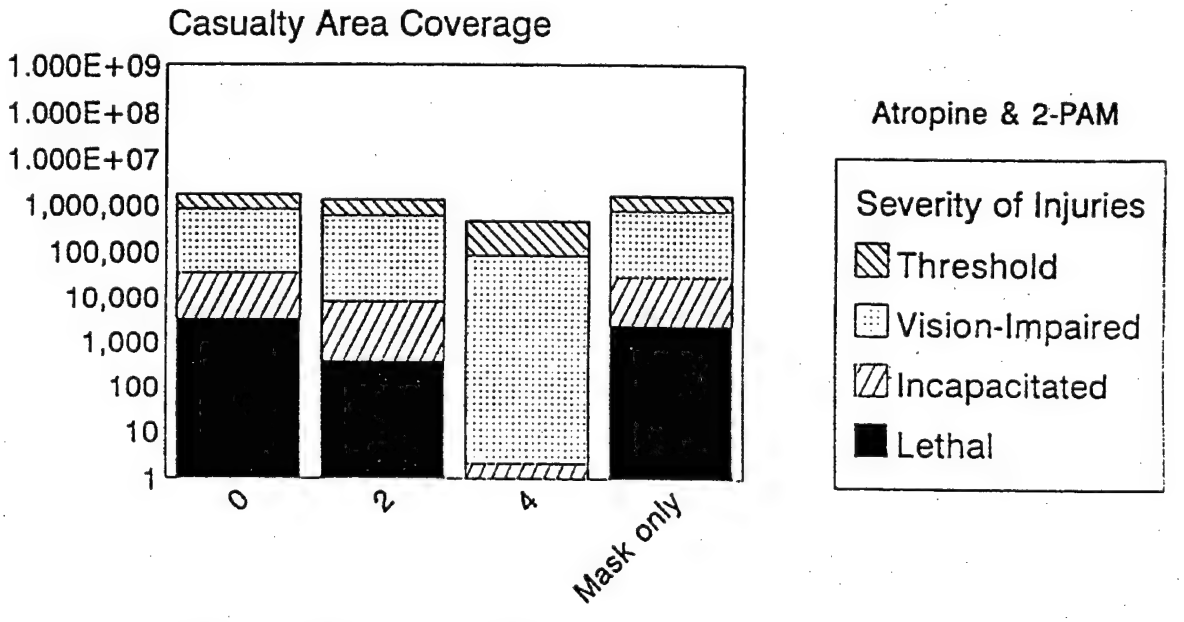
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile with Submunitions
VX



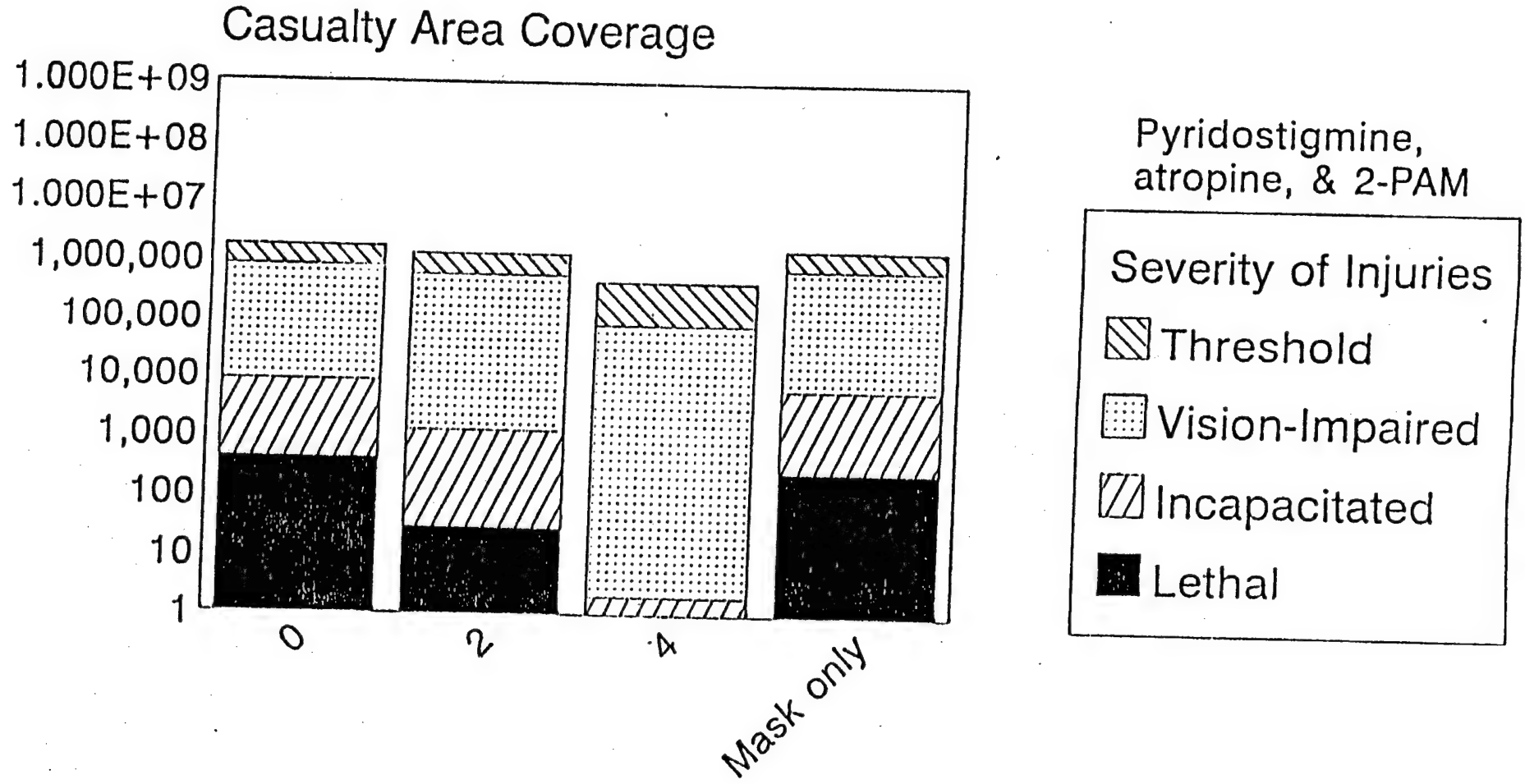
49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
VX



49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile with Submunitions
VX



49°C (120°F) 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE

Sarin (GB)

Tactical Ballistic Missile - Sarin (GB)

An explosive release tactical ballistic missile warhead filled with sarin was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The agent was released from the tactical ballistic missile using either a radar or a laser proximity fuse at a height of 15 meters. Before the Persian Gulf War, use of an explosive release for a bulk ballistic missile warhead was not assessed as being a likely weaponization concept for a variety of reasons. However, the widely publicized Iraqi ballistic missile warhead was just such a system. One very important design issue is the fuzing. The fuse must fire and cause the warhead to function close to the ground (or the agent will evaporate before reaching the ground) but before the warhead buries itself on impact with the ground with velocities greater than the speed of sound.

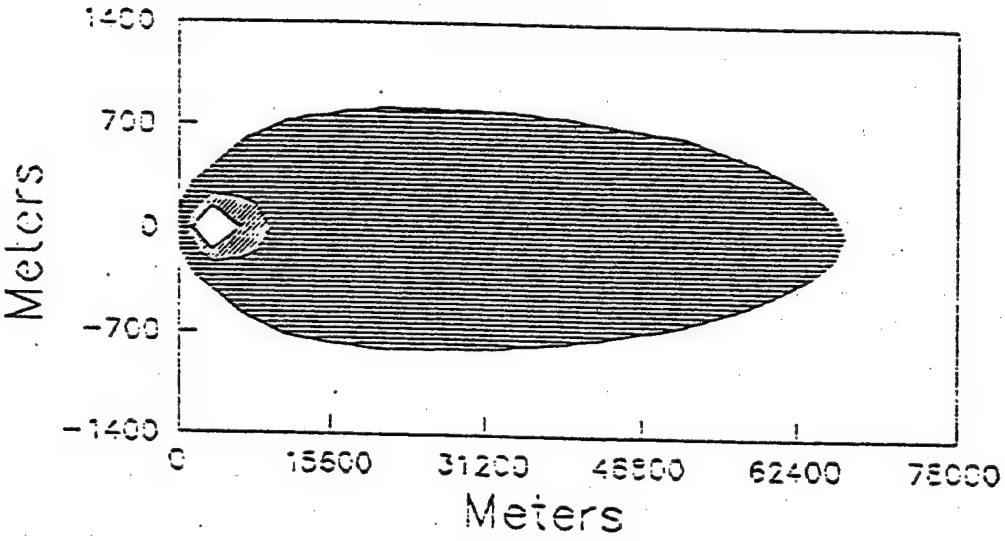
The peak liquid deposition from the attacks demonstrate another potential problem with this type of design. The peak deposition approaches nearly 100 grams/square meter for the 1.5 meter/second windspeed and the 3 meters/second while the 6 meter/second case is still substantially above 10 grams/square meter. These very high peak depositions are matched with deposition area coverages just above 0.1 square kilometers for the 1.5 meters/second and 6 meters/second cases. The 3 meters/second windspeed will produce just above 1 square kilometer.

The combination of large contamination densities and larger median droplet sizes (than typical of explosive munitions) results in concentrations which do not drop below significant levels for more than 2 hours and less than 1.5 days for the low temperature, low windspeed case; between 20 minutes and 1 hours in the moderate temperature, moderate windspeed case; and between 2.5 minutes and 20 minutes for the high temperature, high windspeed case.

The peak dosage is between 1,000 and 10,000 milligram-minutes/cubic meters for the inversion stability conditions with low temperature and windspeed while the Pasquill stability category D, moderate temperature, and moderate windspeed condition as well as the Pasquill stability condition B, high temperature, and high windspeed conditions result in peak dosage values approximately of 1,000 milligram-minutes/cubic meters.

Unprotected lethalities for the low temperature case was approximately 1 square kilometers while the moderate and high temperature cases yielded a likely area coverage of approximately 0.1 square kilometers. There was a three order of magnitude reduction (99.9 per cent reduction) in likely casualty area coverage if the mask is worn. An important characteristic of a sarin attack is that as early as 30 seconds after release, the lethal level achieved is nearly identical with remaining unprotected in the high temperature case and there is less than one order of magnitude reduction (or less than a 90 per cent reduction) in the moderate and low temperature cases.

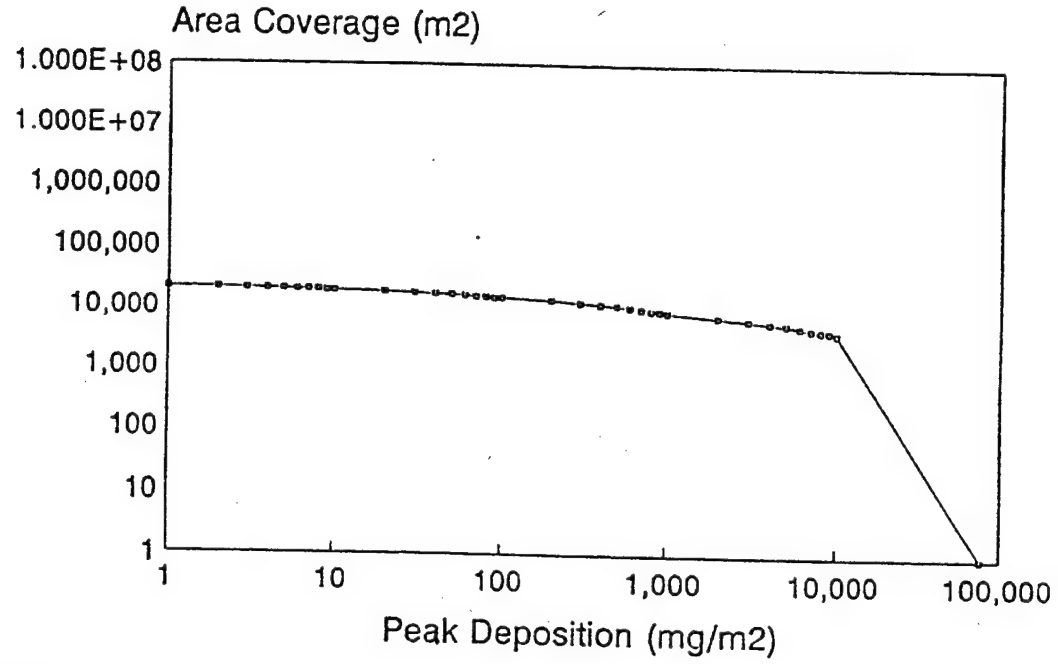
Tactical Ballistic Missile
Sarin (GB)



4oC (40oF)
1.5 m/sec
Stability E

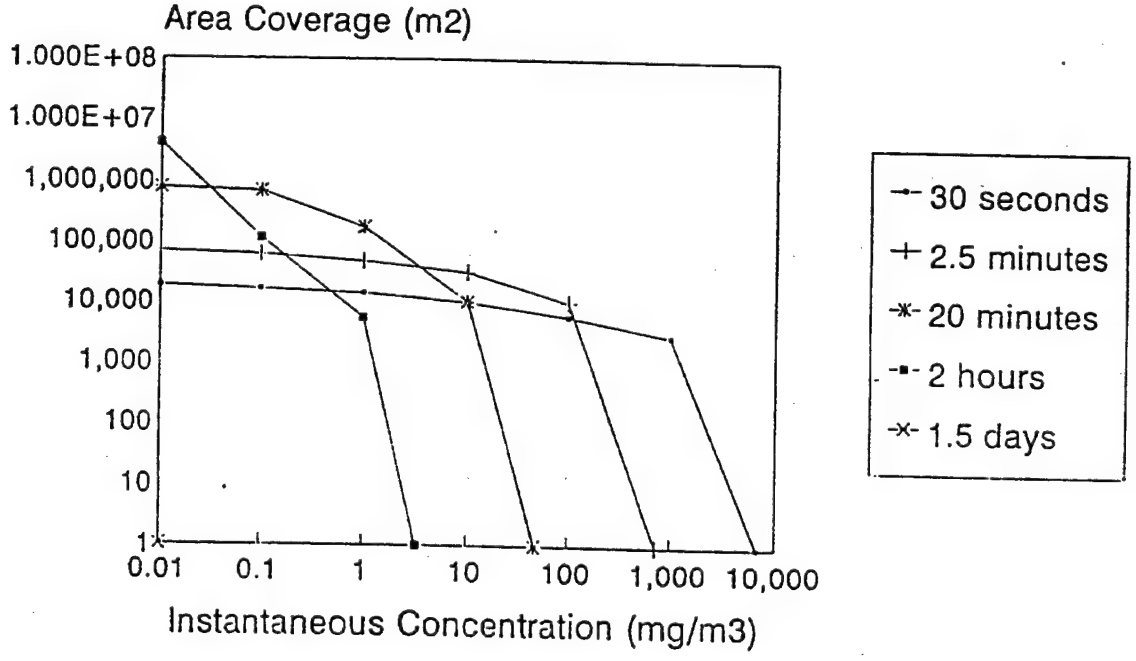
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Sarin (GB)



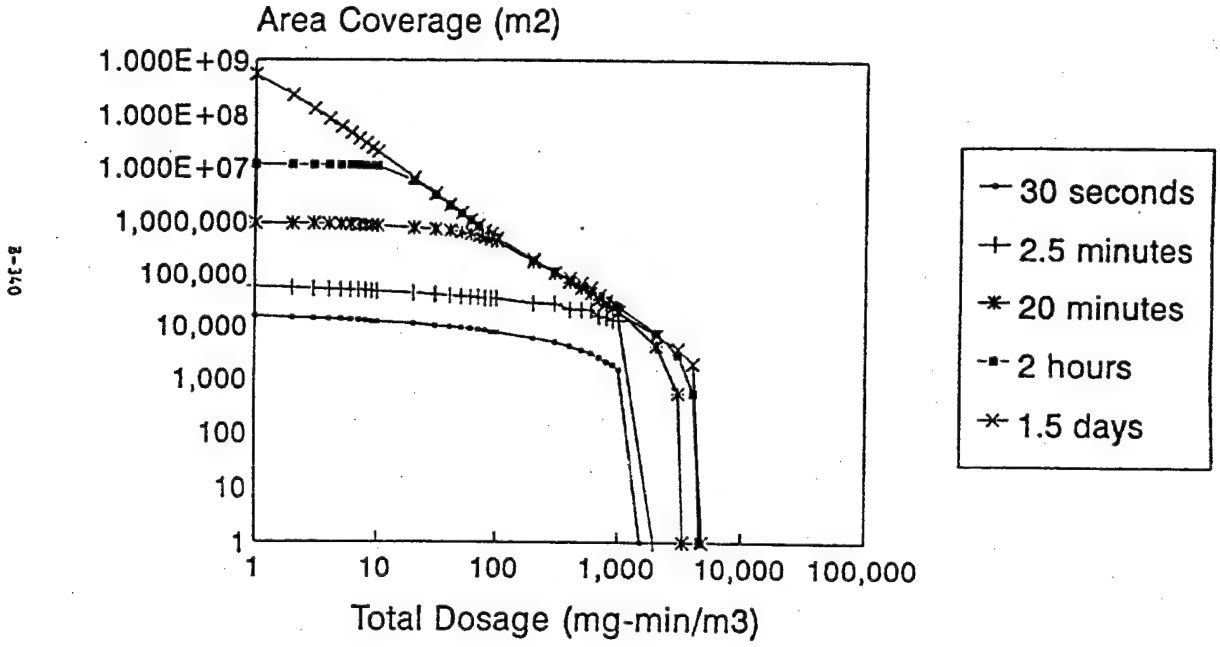
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile
Sarin (GB)



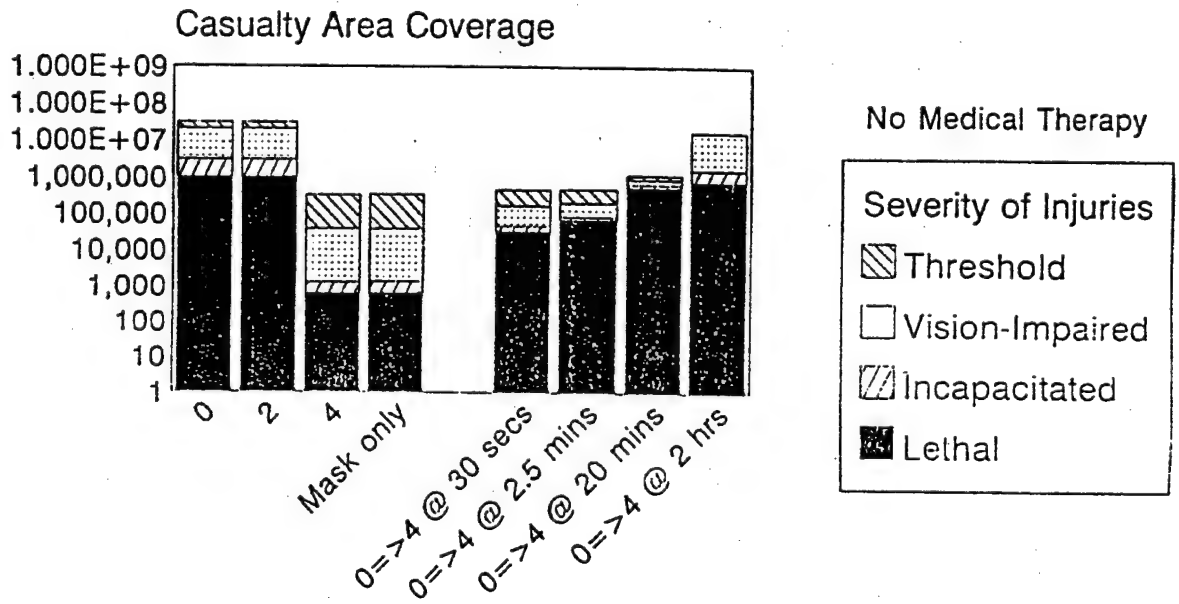
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile
Sarin (GB)



4°C (40°F), 1.5m/sec, stability E

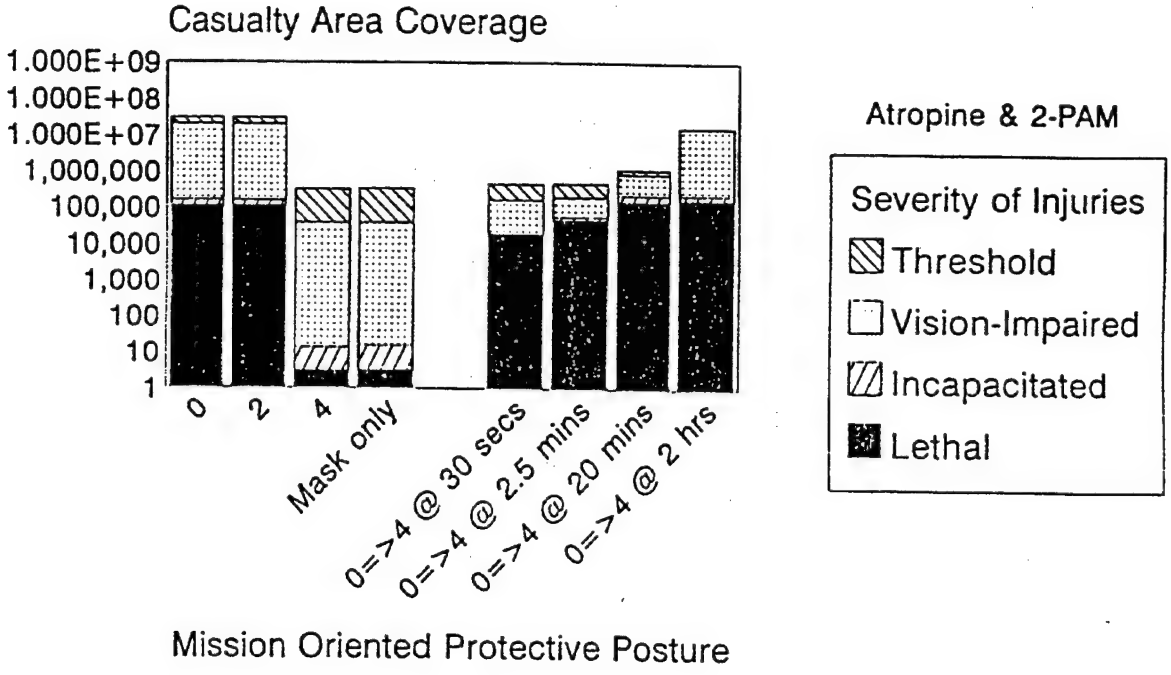
Tactical Ballistic Missile
Sarin (GB)



Mission Oriented Protective Posture

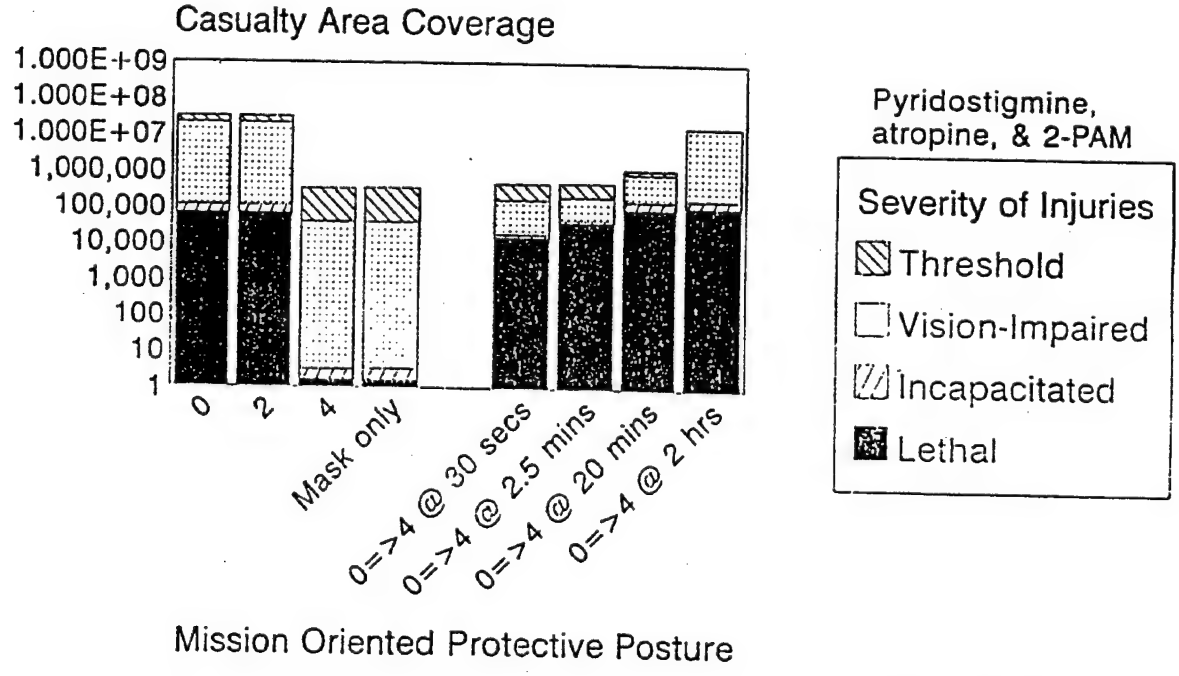
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile
Sarin (GB)

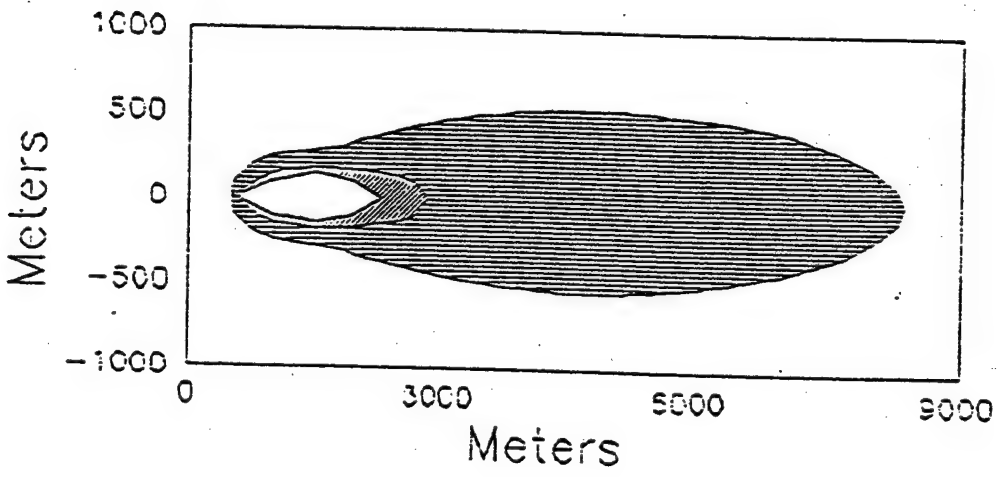


4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile
Sarin (GB)



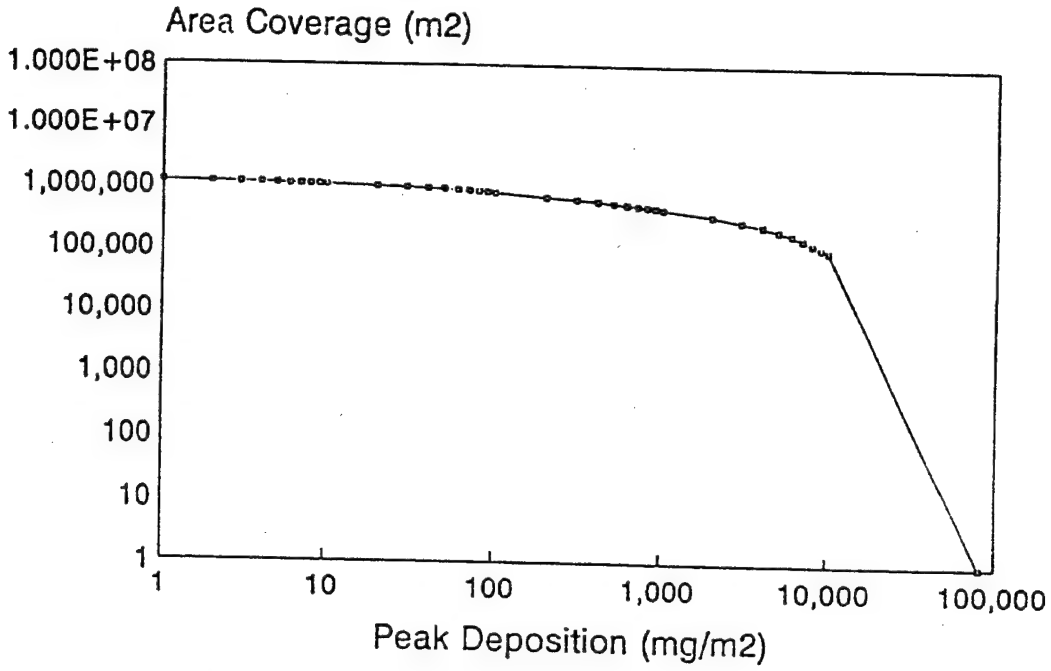
Tactical Ballistic Missile
Sarin (GB)



25oC (77oF)
3 m/sec
Stability D

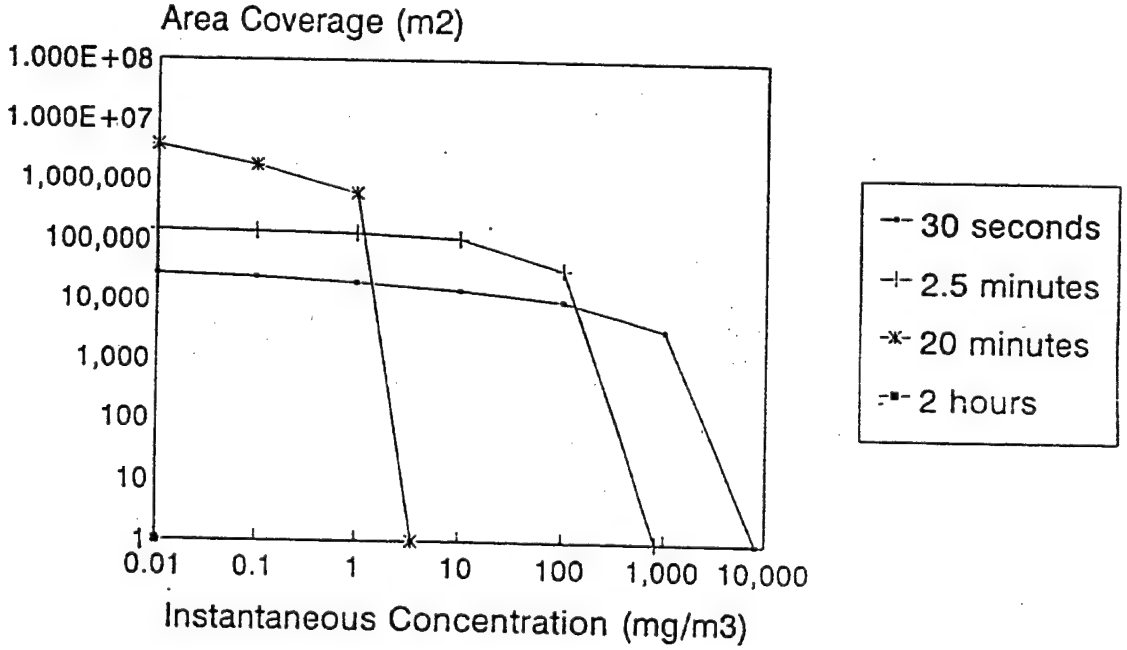
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Sarin (GB)



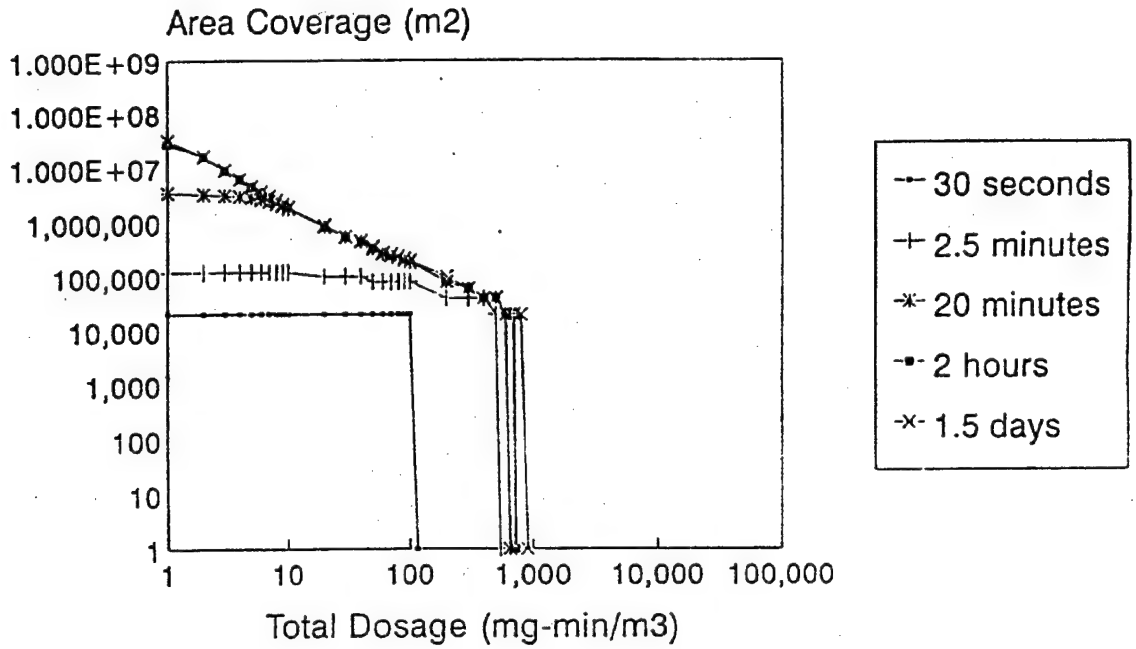
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Sarin (GB)



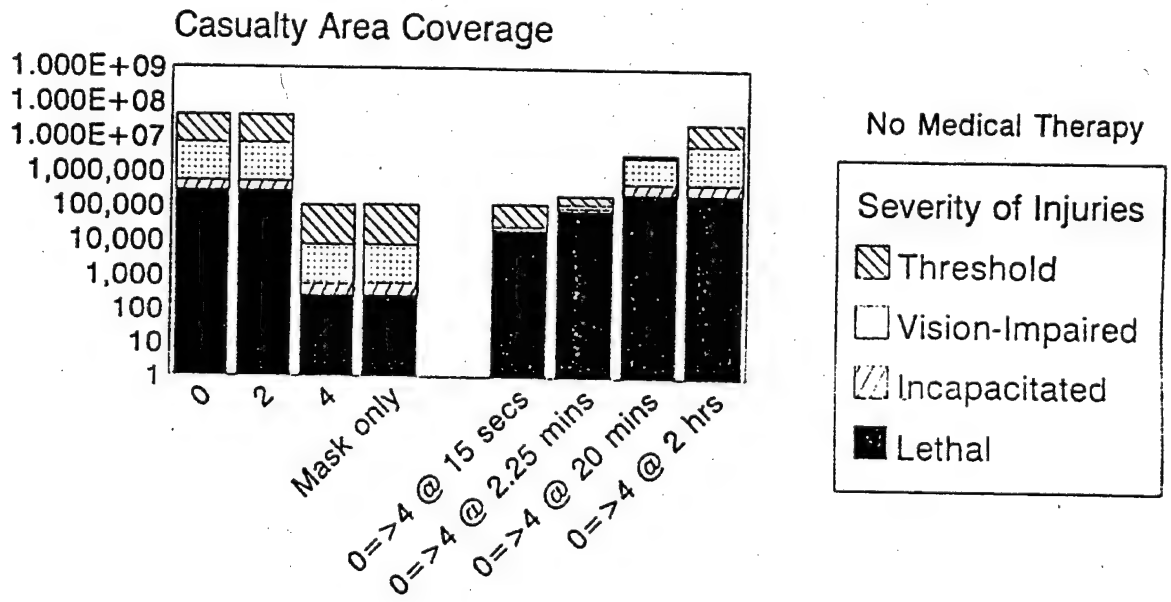
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Sarin (GB)



25°C (77°F), 3m/sec, stability D

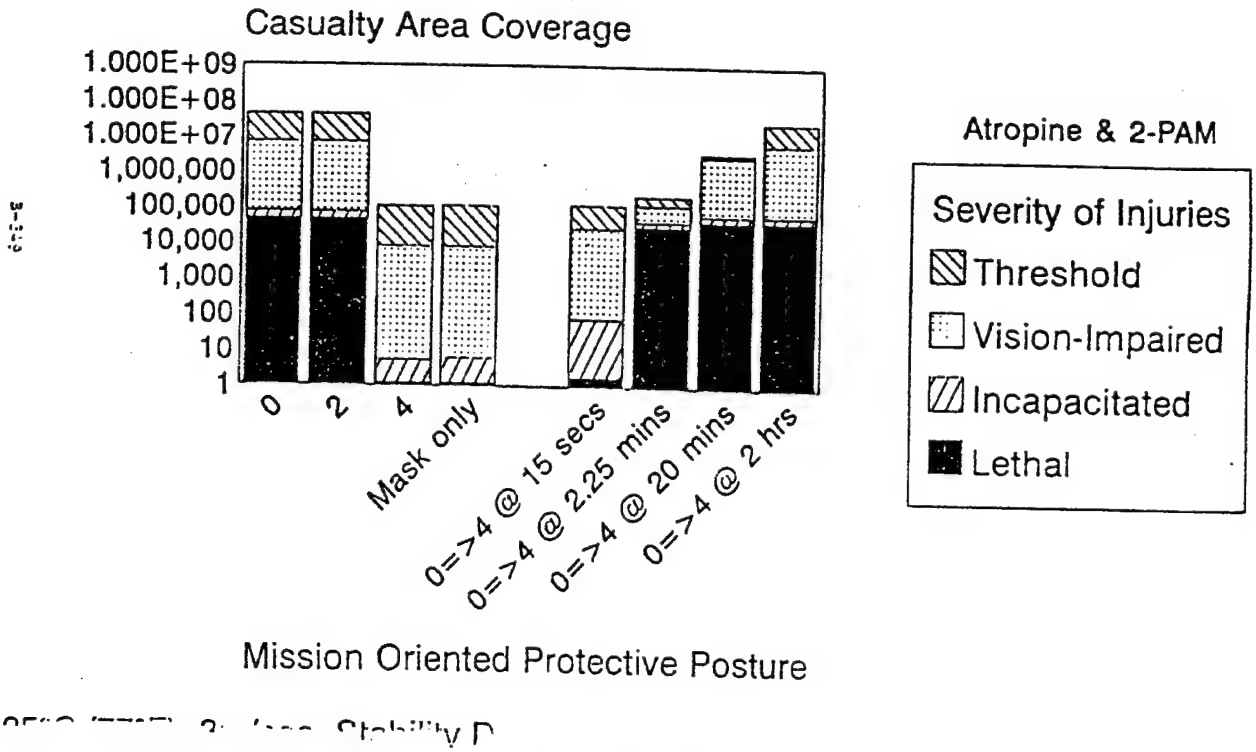
Tactical Ballistic Missile
Sarin (GB)



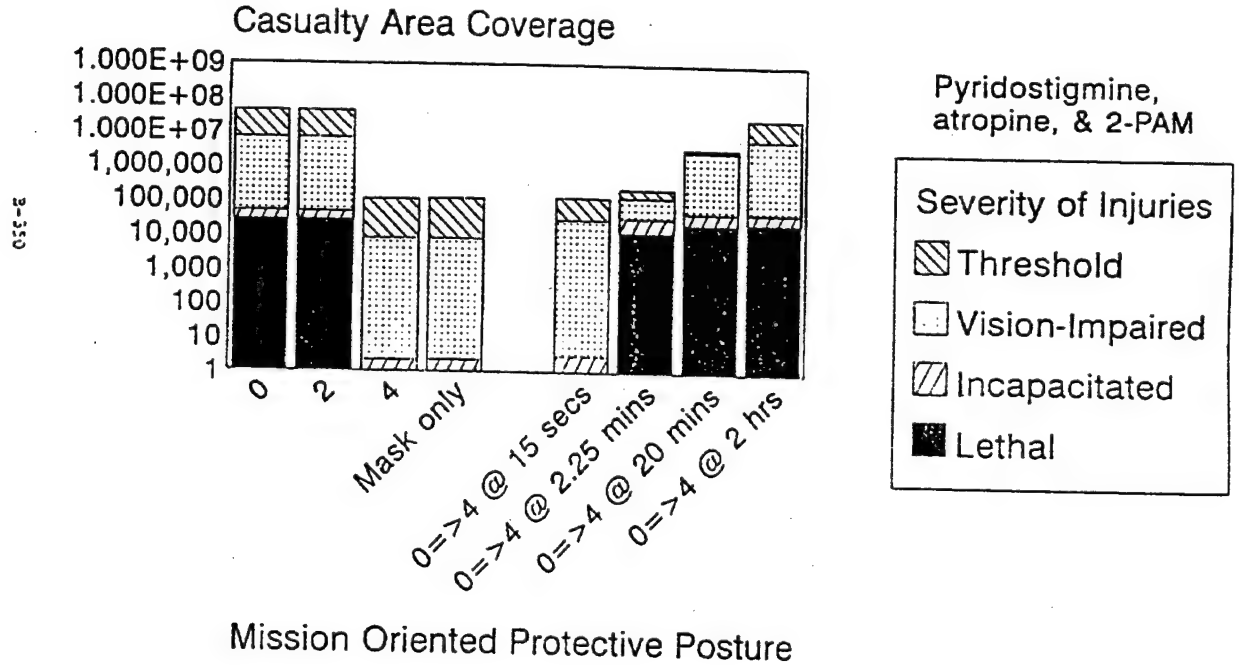
Mission Oriented Protective Posture

25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile
Sarin (GB)

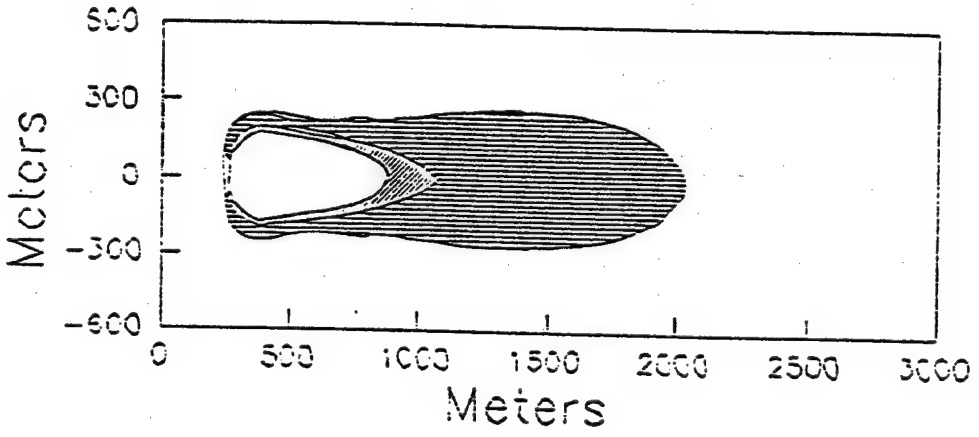


Tactical Ballistic Missile
Sarin (GB)



25°C (77°F), 3m/sec, Stability D

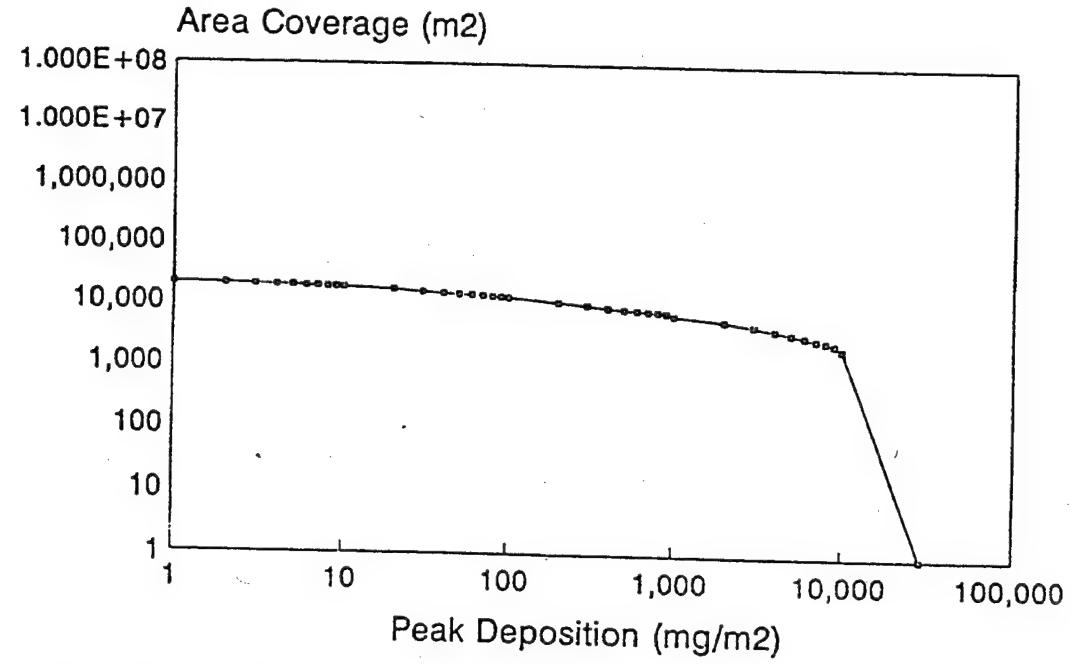
Tactical Ballistic Missile
Sarin (GB)



49oC (120oF)
6 m/sec
Stability B

Visually Impaired
Incapacitated
Lethal

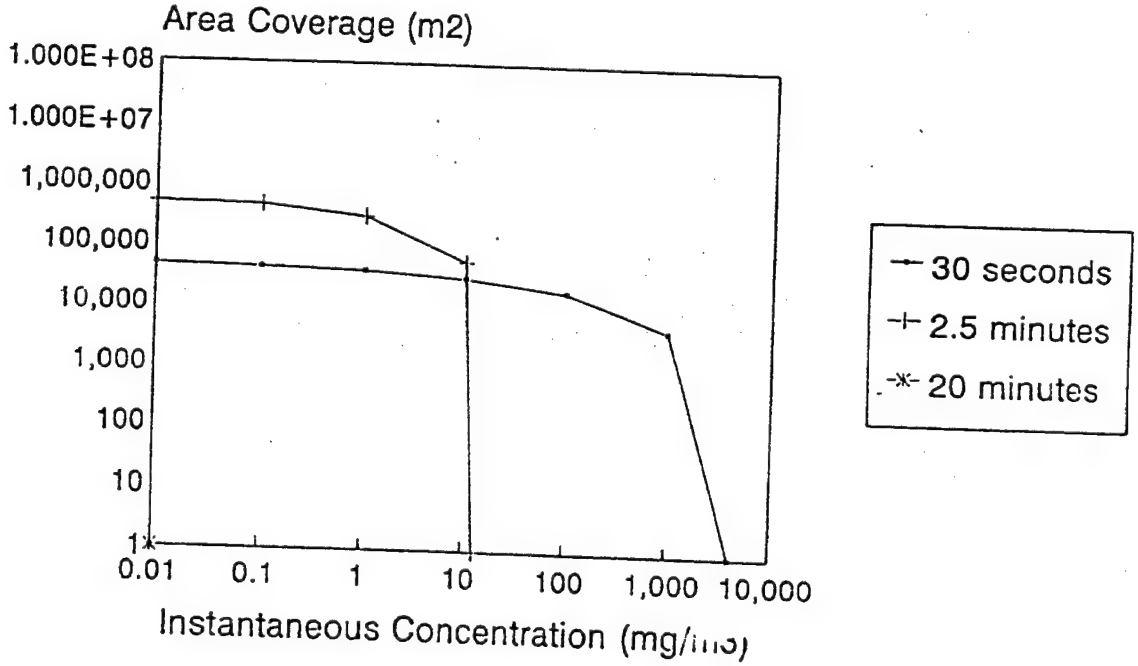
Tactical Ballistic Missile
Sarin (GB)



3-332

49°C (120°F), 6m/sec, stability B

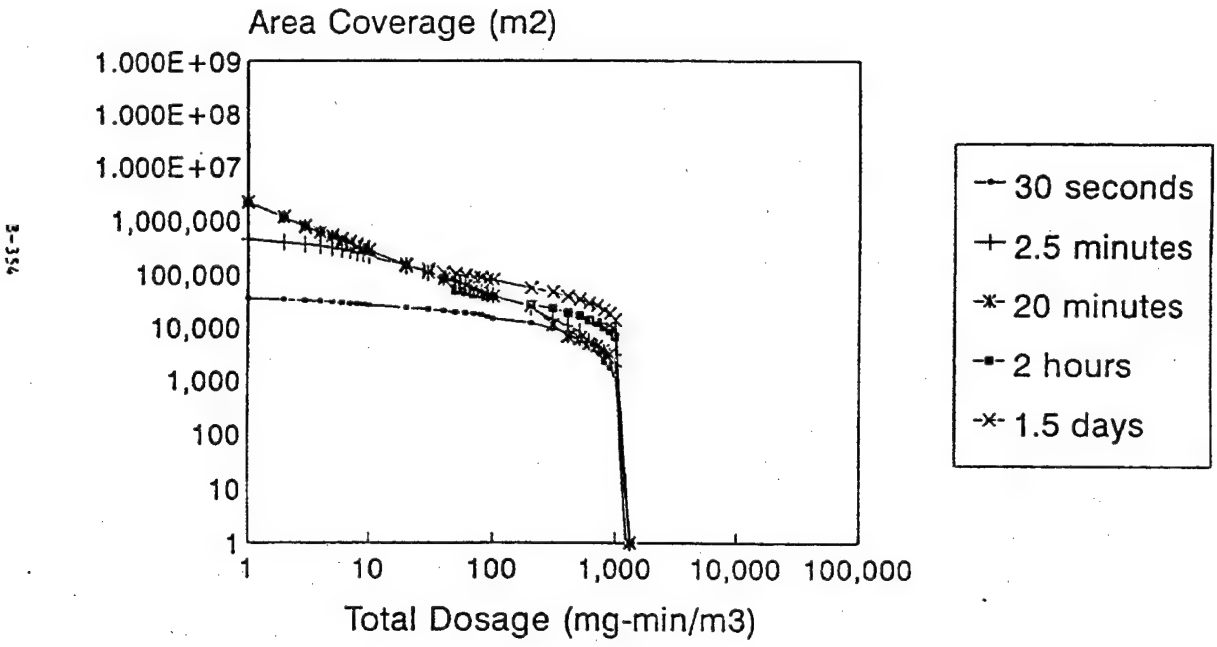
Tactical Ballistic Missile
Sarin (GB)



3-333

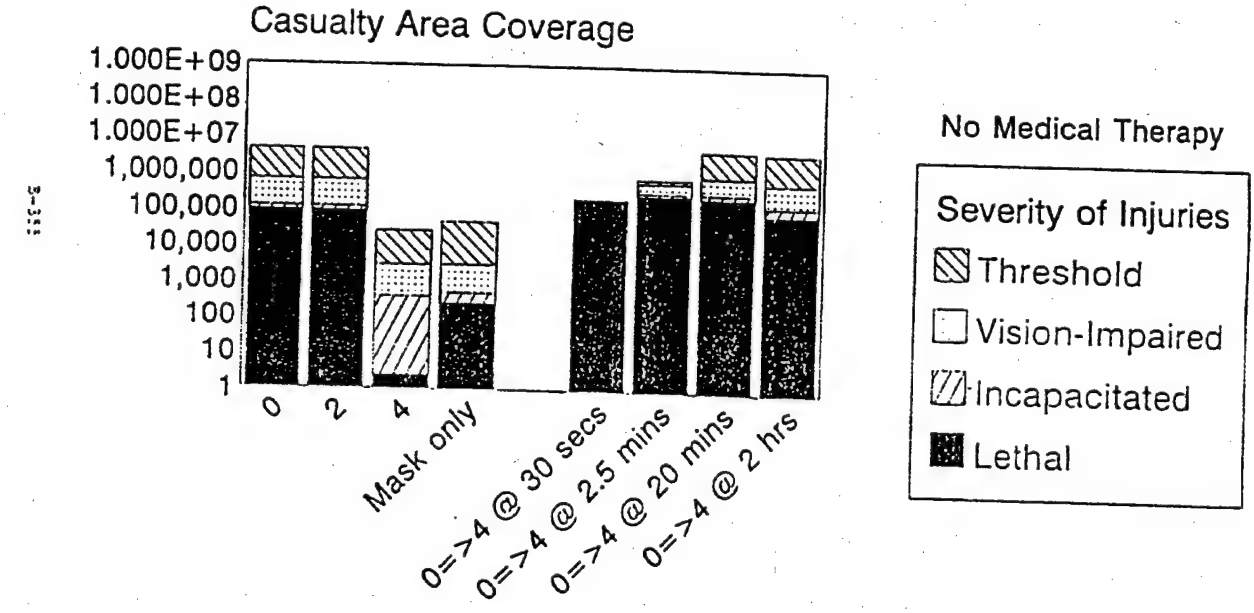
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile
Sarin (GB)



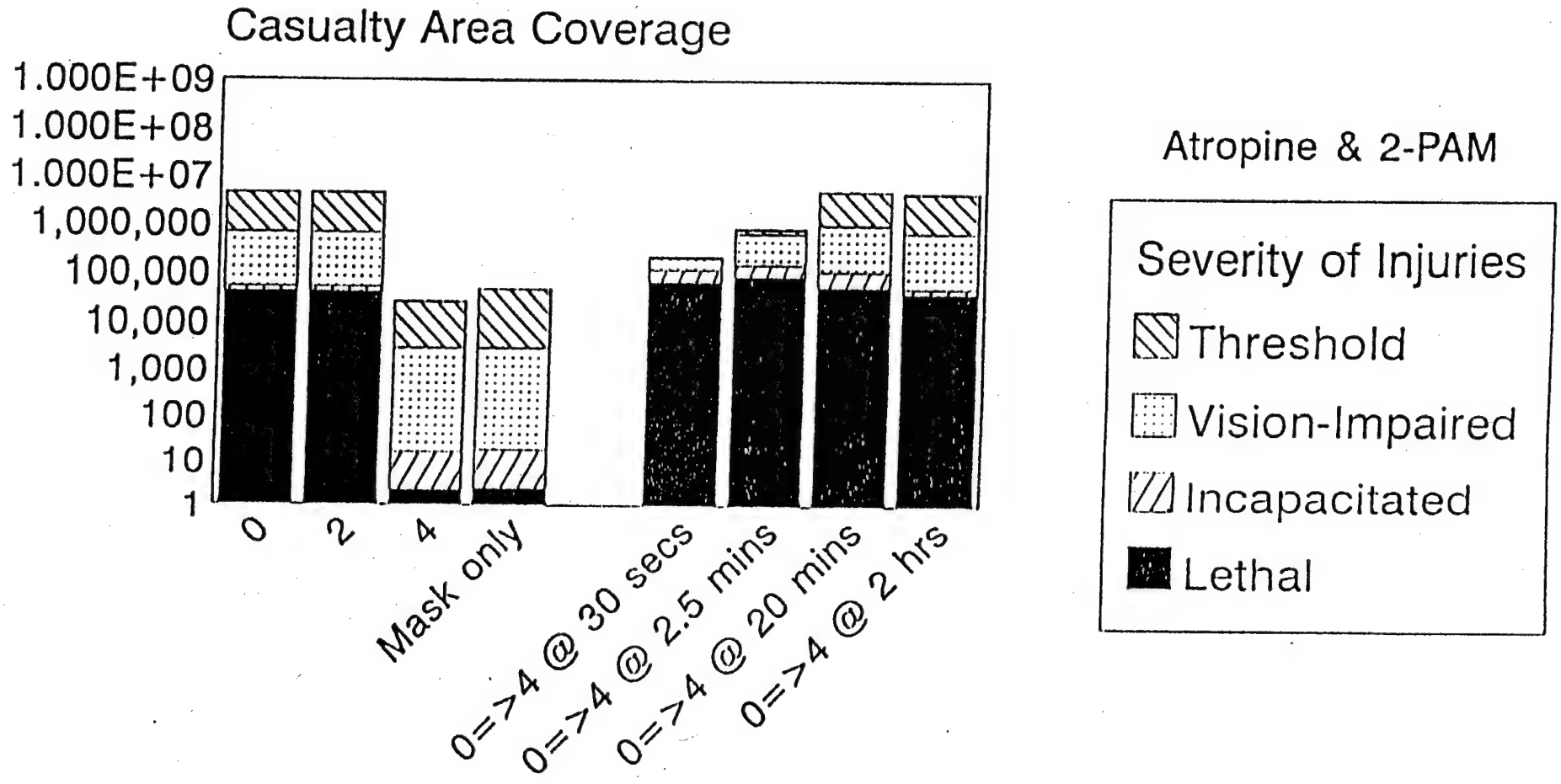
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile
Sarin (GB)



Tactical Ballistic Missile

Sarin (GB)



49°C (120°F), 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE

Thickened Soman (TGD)

Tactical Ballistic Missile - Thickened Soman (TGD)

A bulk release tactical ballistic missile warhead filled with over 500 kilograms of thickened soman. The system was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The agent was released from the tactical ballistic missile using either a barometric or a laser proximity fuse at an altitude tuned to the windspeed. The release for the 1.5 meter/second windspeed case was 900 meters. The release for the 3 meter/second windspeed case was 750 meters. The release for the 6 meter/second windspeed case was 600 meters. The line of agent produced by the release was 300 meters long and the median droplet diameter was 800 microns.

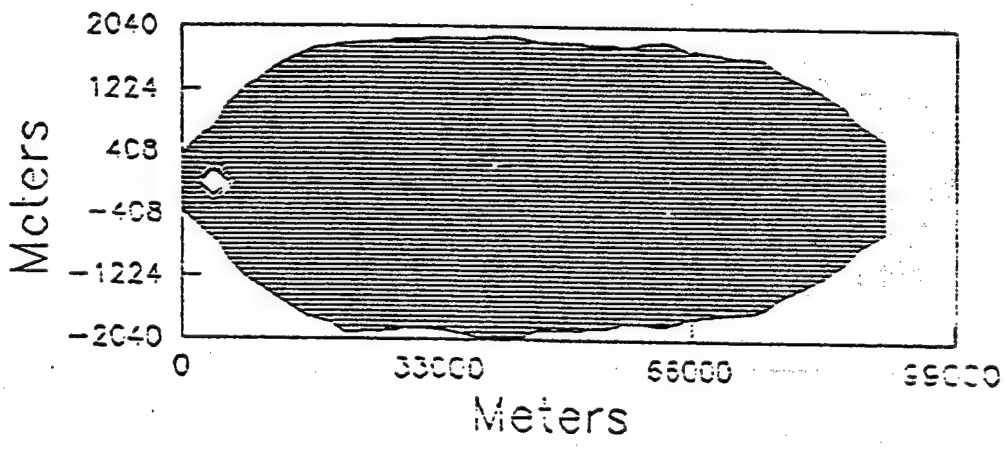
The peak liquid deposition from the attacks was less than 10 grams/square meter for the 1.5 meter/second windspeed and the 3 meter/second windspeed, but less than 1 grams/square meter for the 6 meter/second windspeed.

The concentration area coverage curves show that the peak concentration values were reached very early as represented by the results at 1 hour after the attack in the low temperature case and 3 minutes after the attack in the moderate and high temperature cases. It is useful to note that no concentration was generated at 1 minute after the munition function. The concentration across the target area drops below significant levels between 1 hour and 16 hours for the low temperature, low windspeed case and the moderate temperature, moderate windspeed case. The concentration across the target area drops below significant levels between 3 minutes and 1 hour for the high temperature, high windspeed case.

The dosage area coverage curves achieved peak dosage values of approximately 1,000 milligram-minutes/cubic meter for the low temperature and moderate temperature cases, but only achieves a peak value of less than 100 milligram-minutes/cubic meter for the high temperature case.

The casualty area coverage curves show the likely lethal area coverage of approximately 1 square kilometer for unprotected personnel in the low and moderate temperature conditions while the high temperature case achieved less than a 0.1 square kilometer area. Wearing full protective equipment (MOPP 4) reduced the likely lethal area between two and three orders of magnitude (or between 99 and 99.9 per cent reduction) of lethal area during the low and moderate temperature values. For the high temperature conditions, the lethal area was reduced by over four orders of magnitude (or more than a 99.99 per cent reduction.

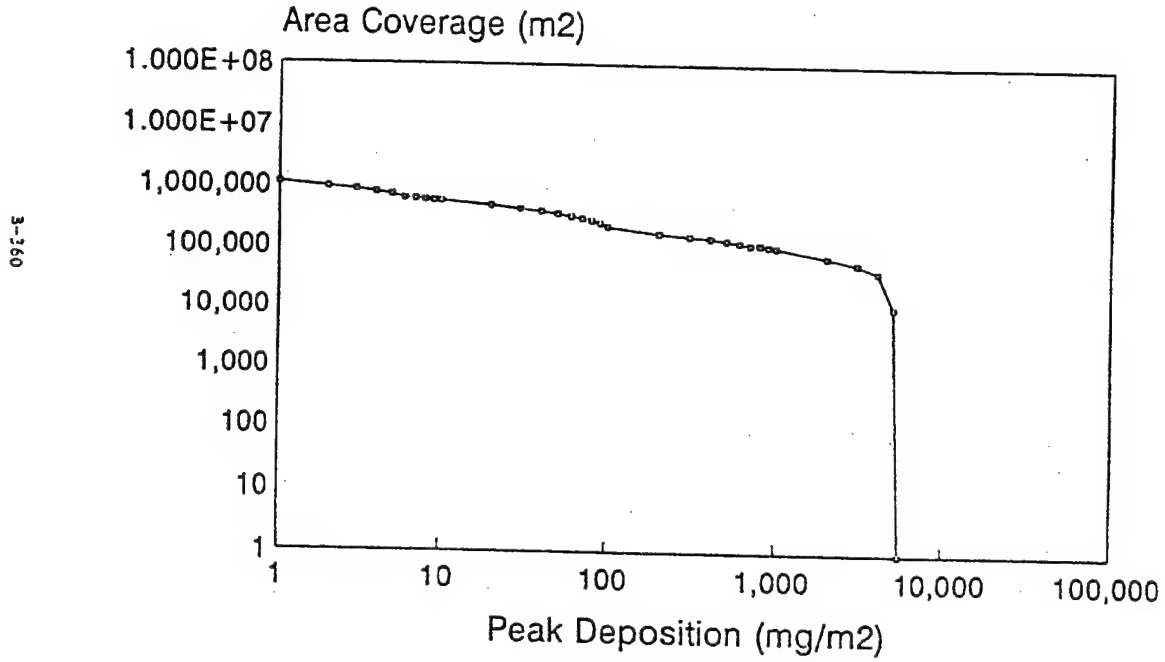
Tactical Ballistic Missile
Thickened Soman (GD)



4oC (40oF)
1.5 m/sec
Stability E

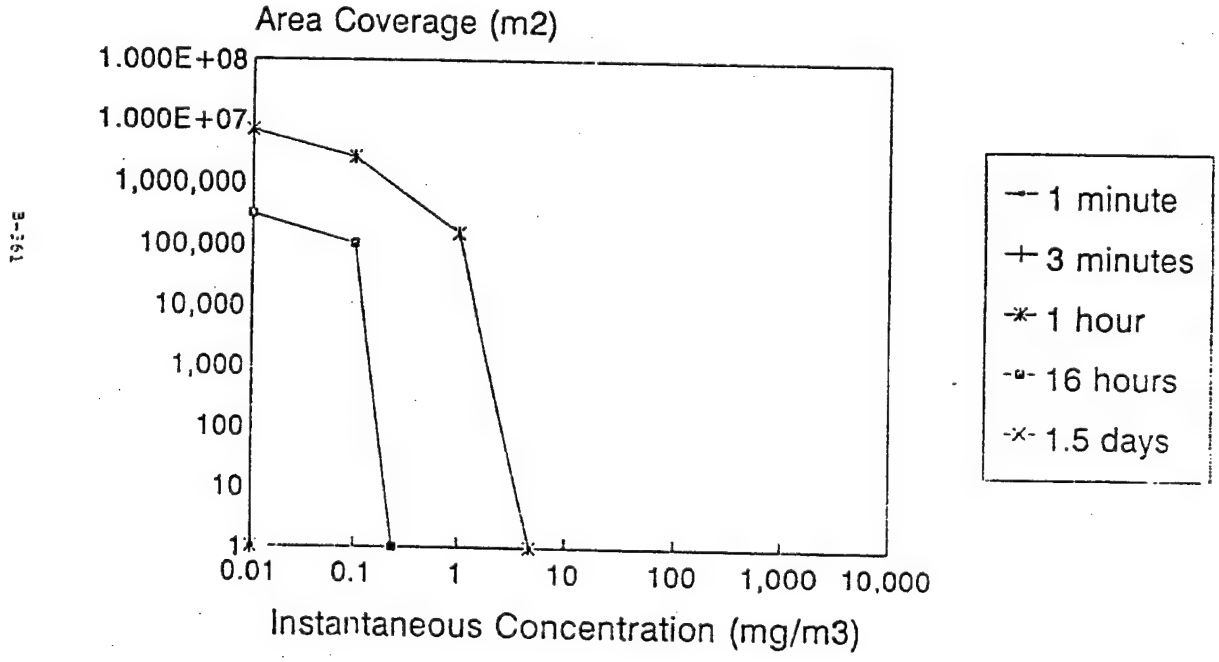
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened Soman (GD)



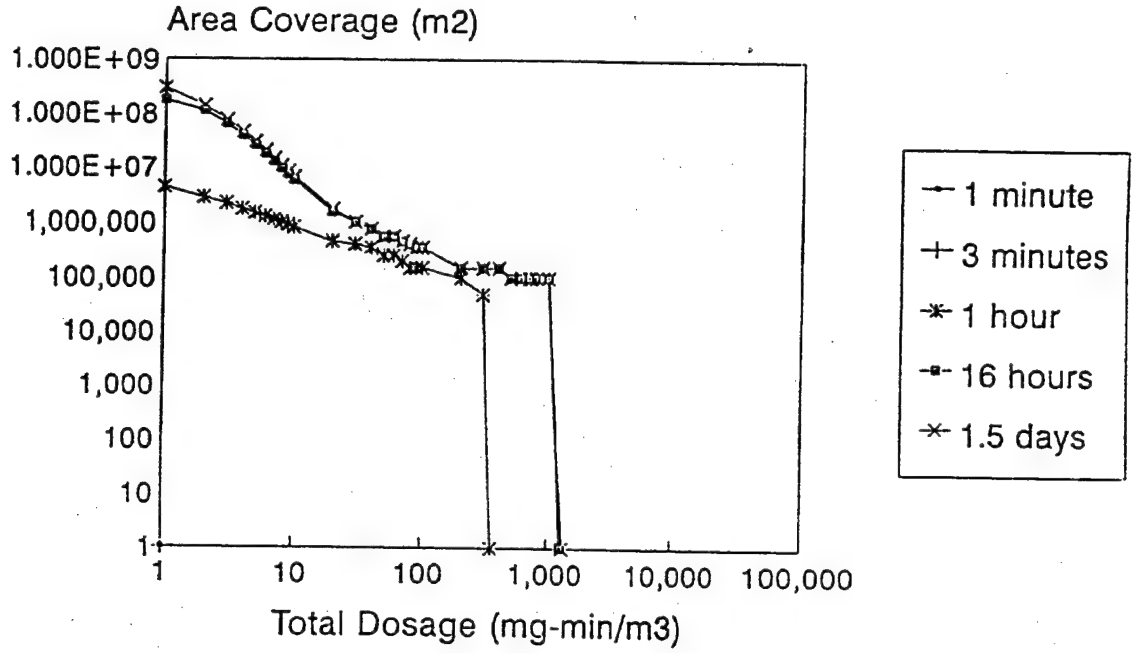
4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile
Thickened Soman (GD)



4°C (40°F), 1.5m/sec, stability E

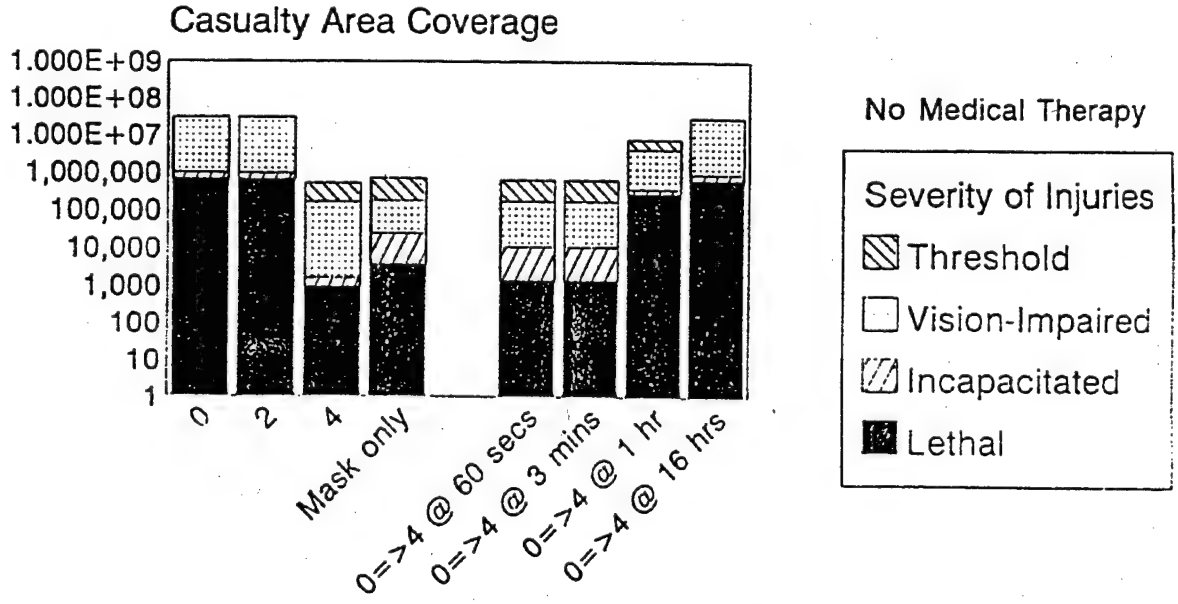
Tactical Ballistic Missile
Thickened Soman (GD)



3-162

4°C (40°F), 1.5m/sec, stability E

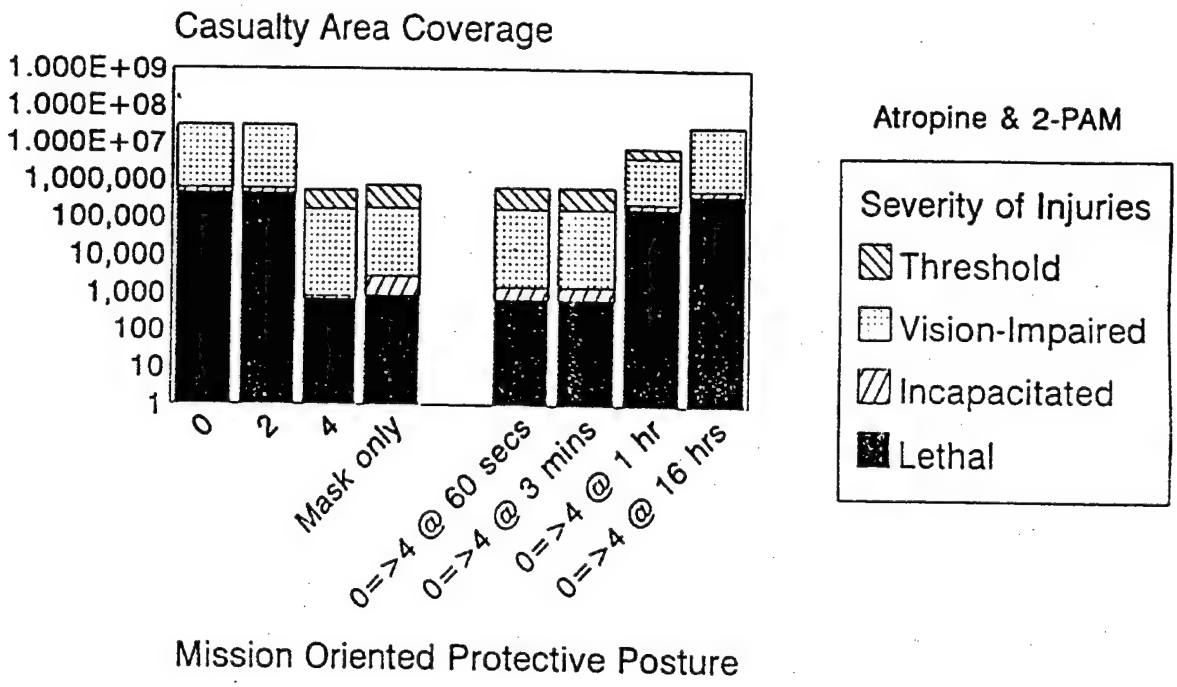
Tactical Ballistic Missile
Thickened Soman (GD)



3-163

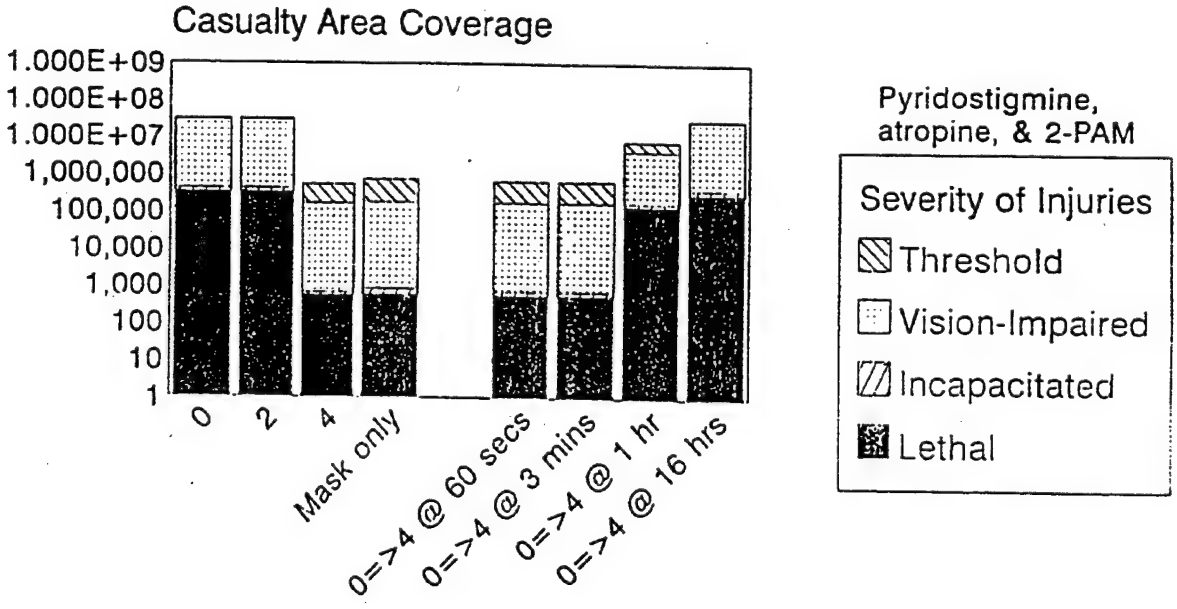
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile
Thickened Soman (GD)



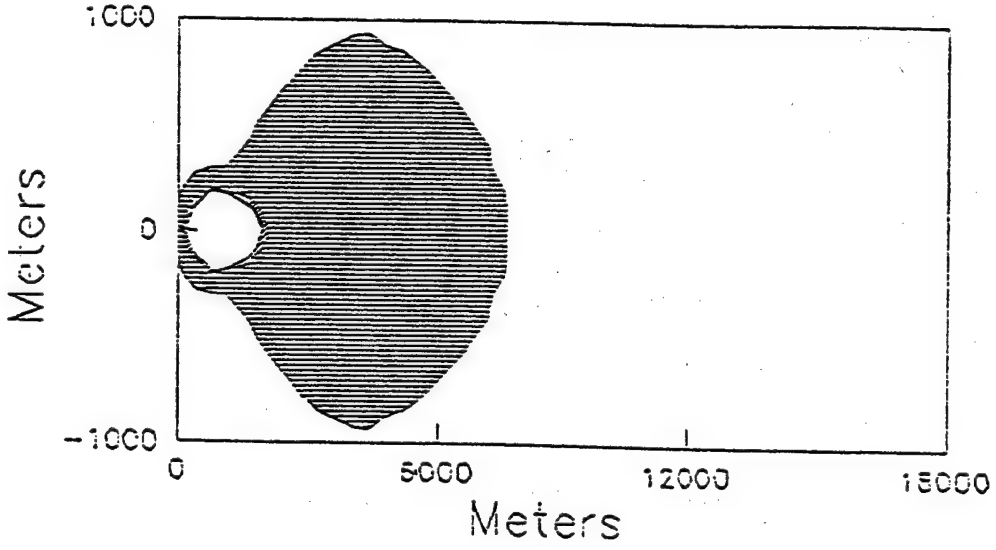
4°C (40°F), 1.5m/sec, Stability E

Tactical Ballistic Missile
Thickened Soman (GD)



(10°F), 1.5m/sec, Stability E

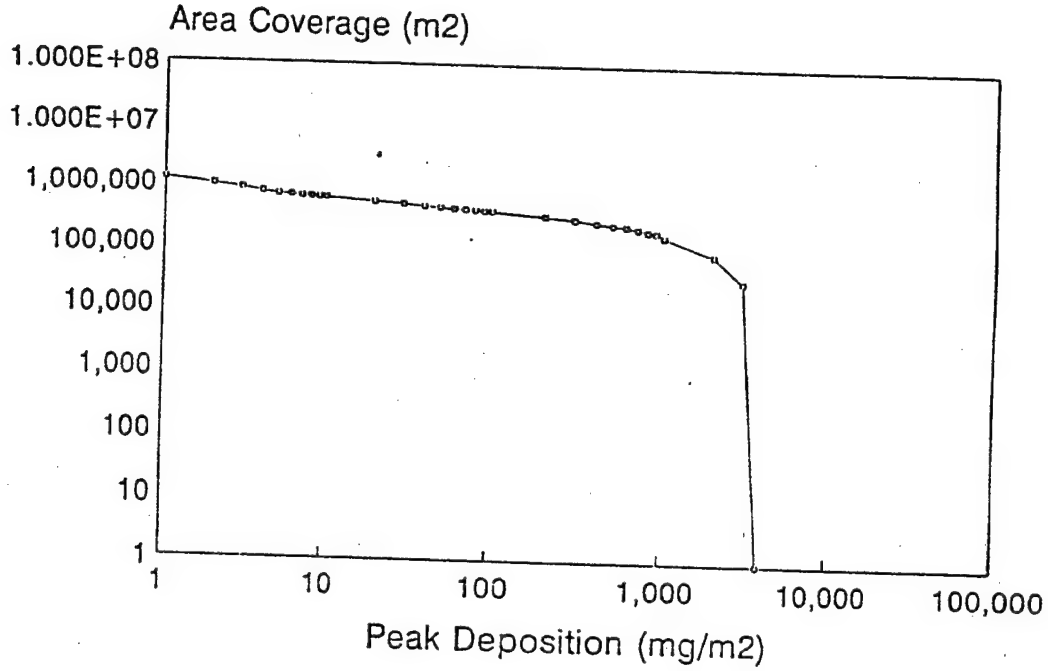
Tactical Ballistic Missile
Thickened Soman (GD)



25oC (77oF)
3 m/sec
Stability D

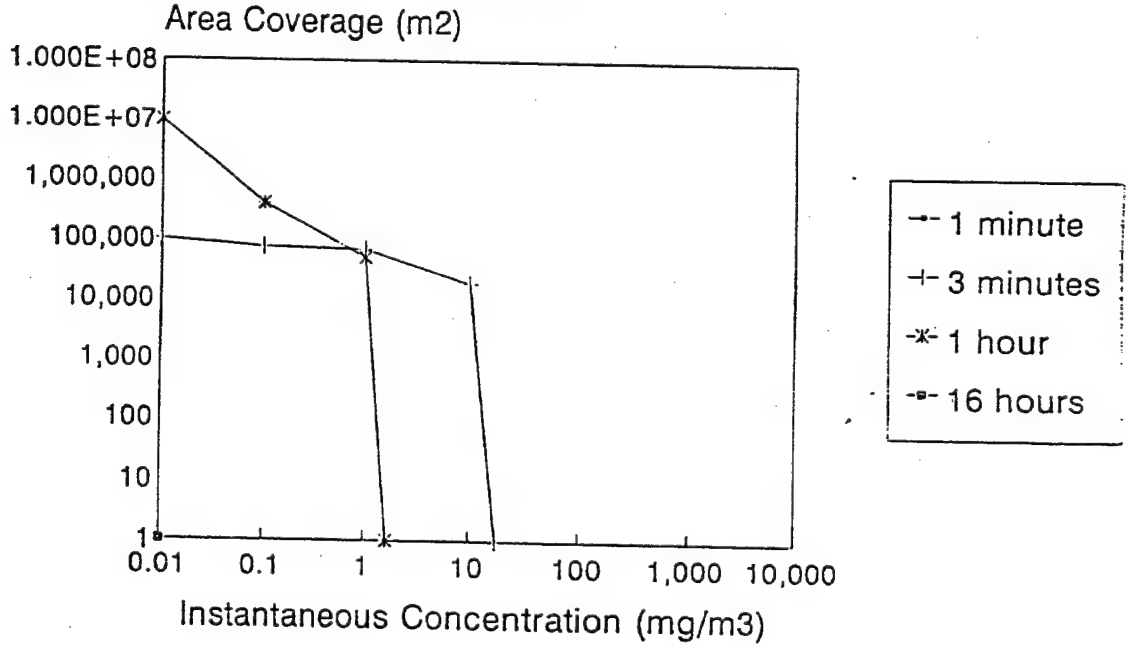
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened Soman (GD)



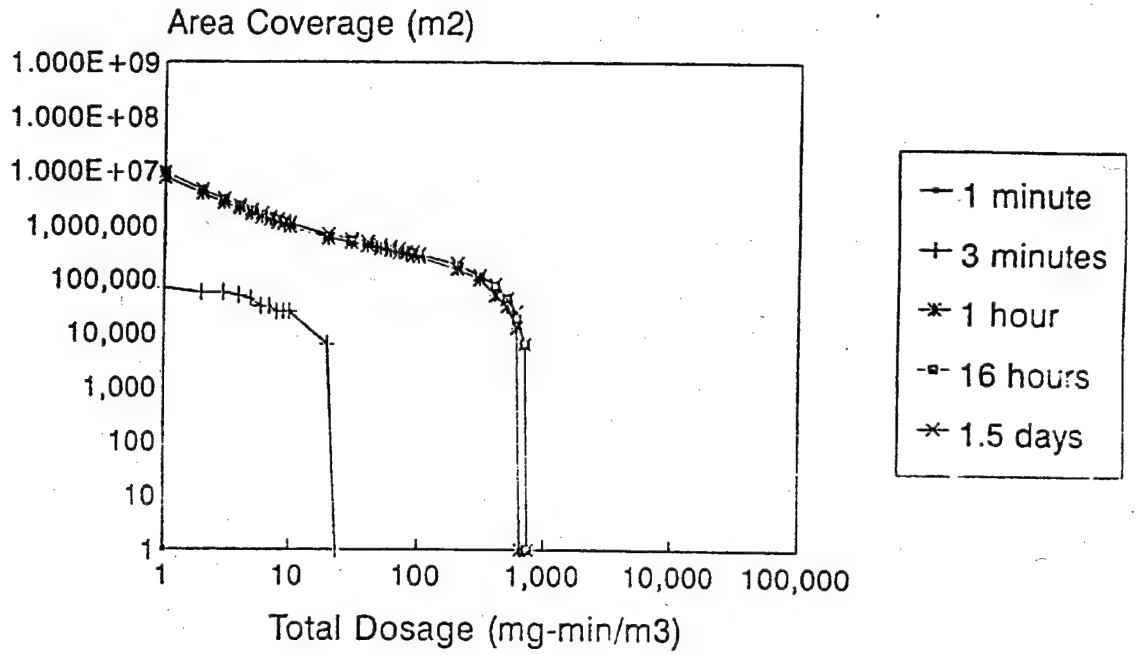
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Thickened Soman (GD)



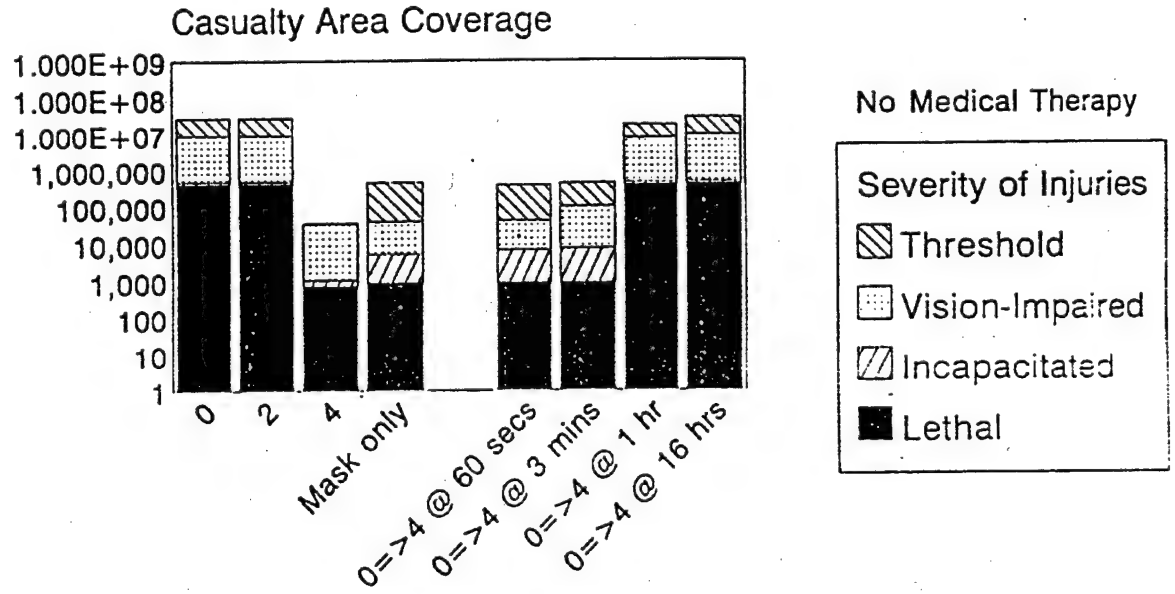
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Thickened Soman (GD)



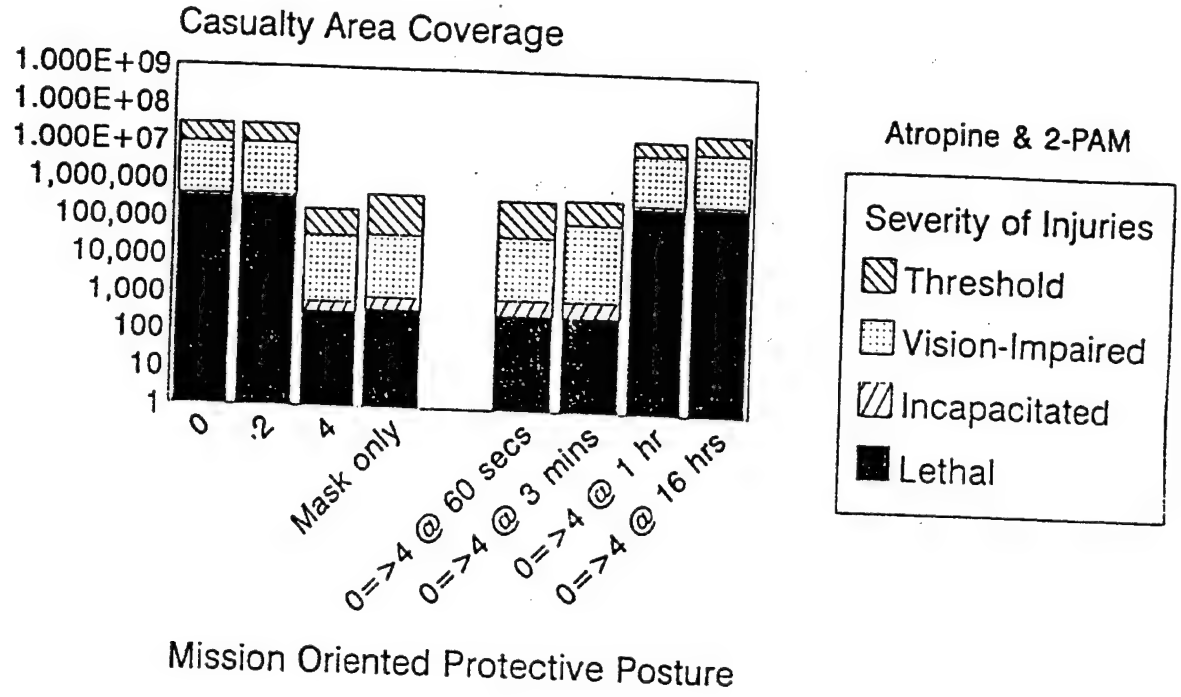
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Thickened Soman (GD)



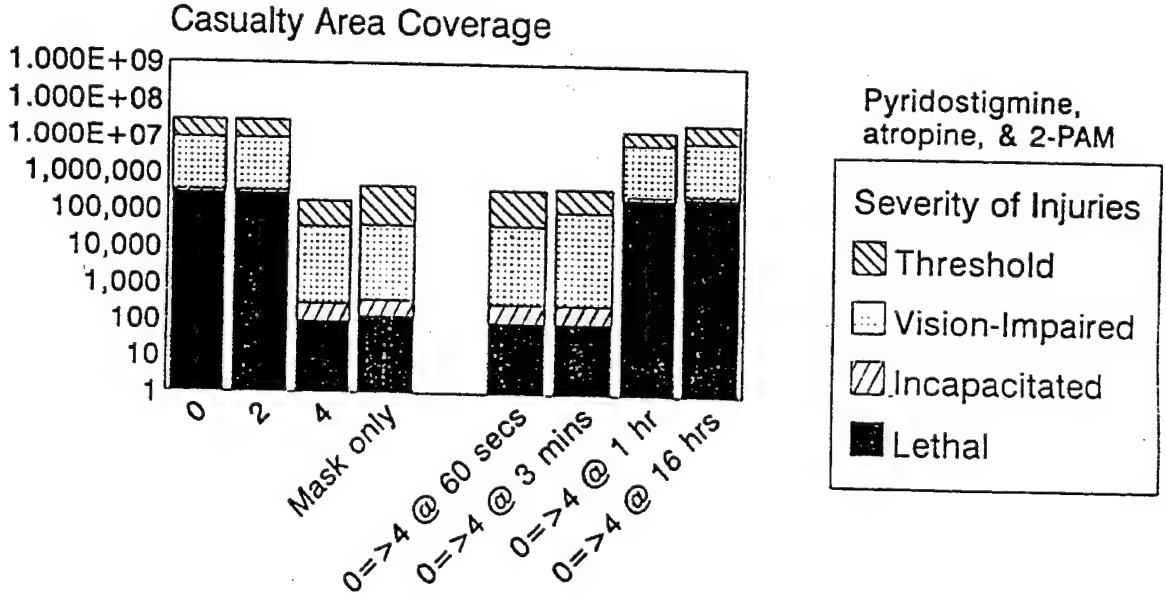
25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile
Thickened Soman (GD)



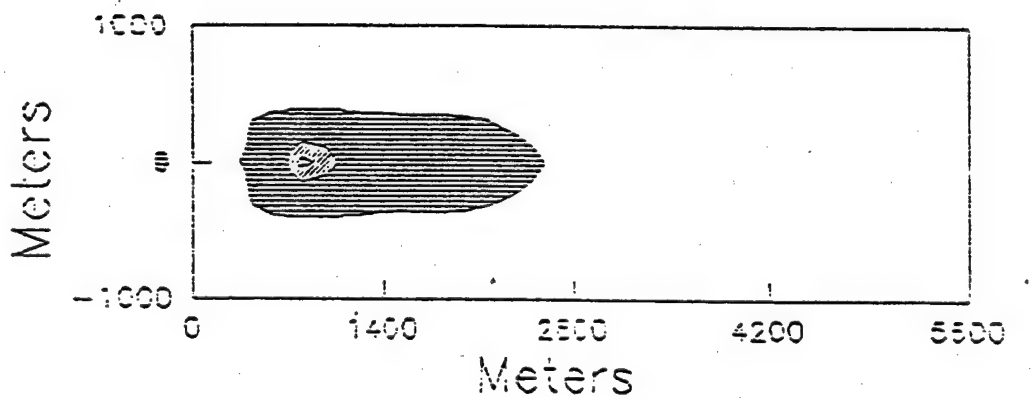
25°C (77°F) 3m/sec, Stability D

Tactical Ballistic Missile
Thickened Soman (GD)



25°C (77°F), 3m/sec, Stability D

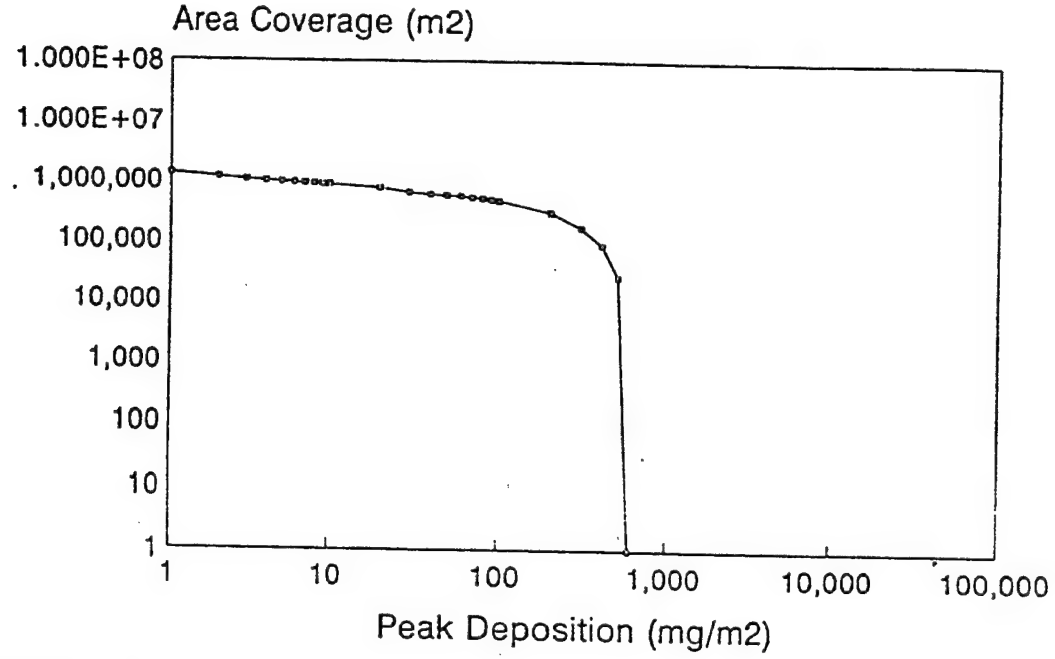
Tactical Ballistic Missile
Thickened Soman (GD)



49oC (120oF)
6 m/sec
Stability B

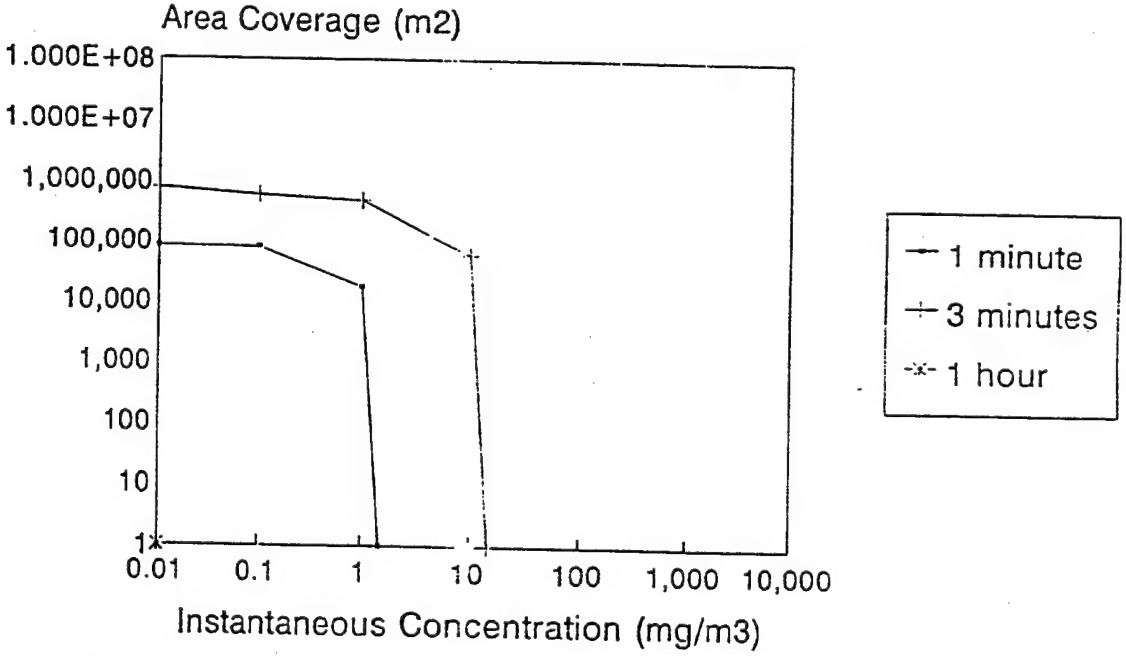
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened Soman (GD)



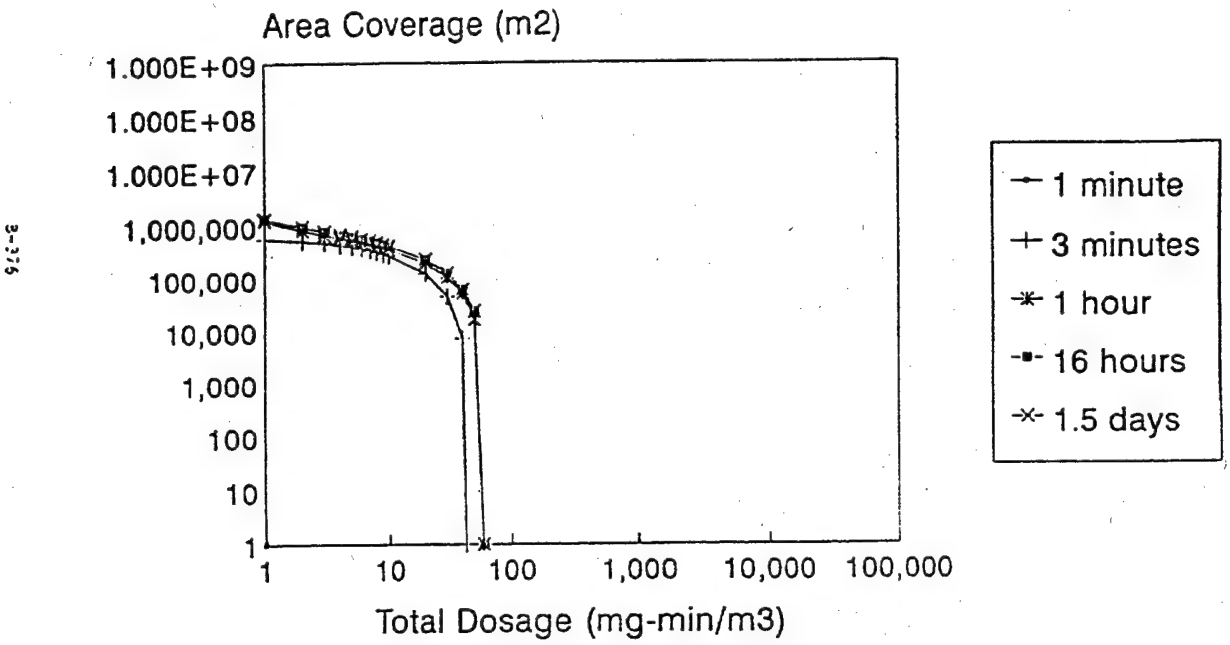
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile
Thickened Soman (GD)



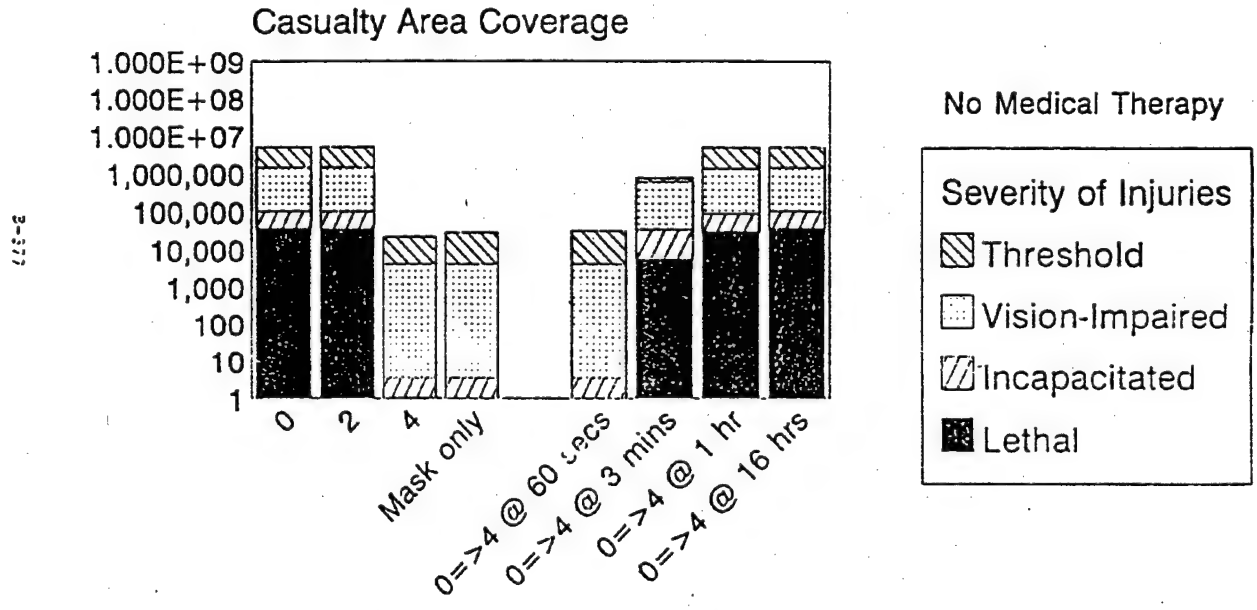
49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile
Thickened Soman (GD)



49°C (120°F), 6m/sec, stability B

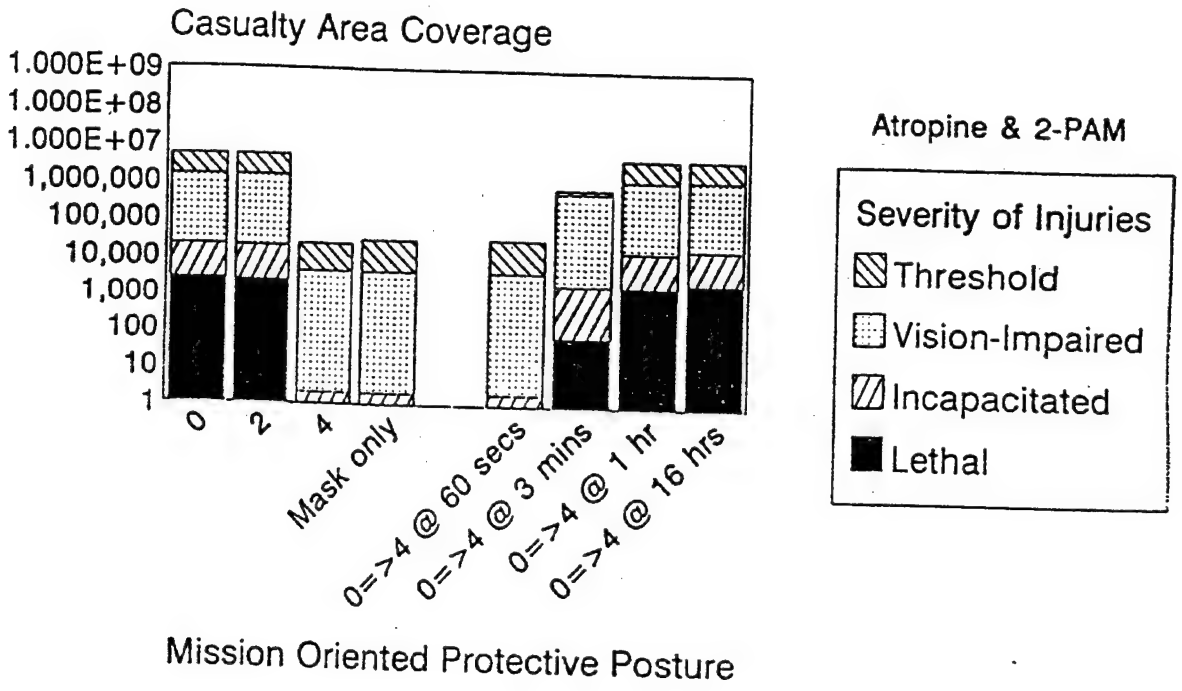
Tactical Ballistic Missile
Thickened Soman (GD)



Mission Oriented Protective Posture

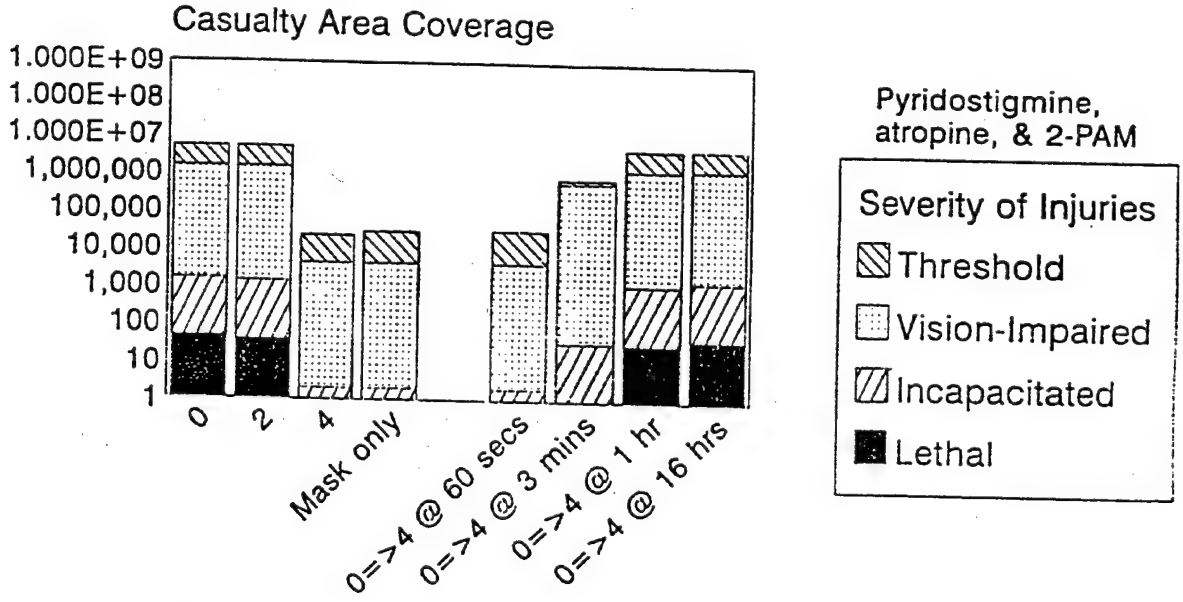
49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile
Thickened Soman (GD)



49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile
Thickened Soman (GD)



49°C (120°F), 6m/sec, Stability B

TACTICAL BALLISTIC MISSILE

Thickened VX (TVX)

Tactical Ballistic Missile - Thickened VX (TVX)

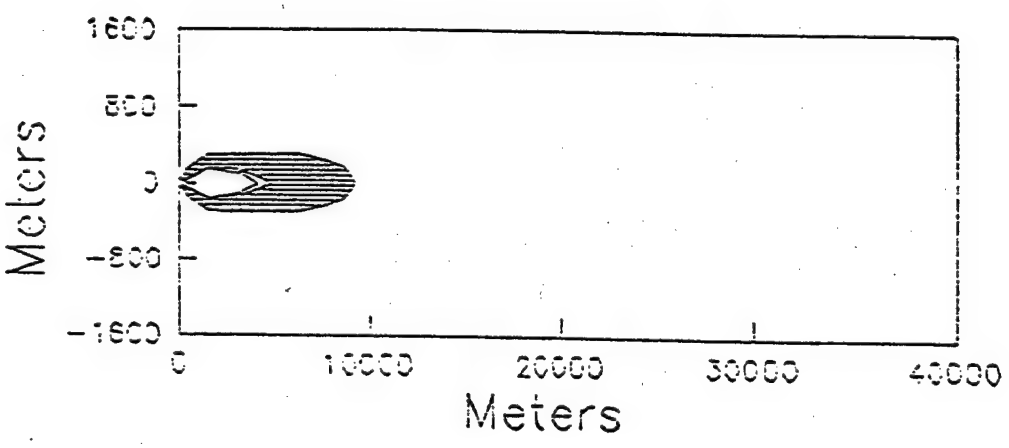
A bulk release tactical ballistic missile warhead filled with sarin was represented for three different combinations of air temperature, windspeed, and atmospheric stability category. The agent was released from the tactical ballistic missile using either a barometric or a laser proximity fuse at an altitude tuned to the windspeed. The release for the 1.5 meter/second windspeed case was 600 meters. The release for the 3 meter/second windspeed case was 350 meters. The release for the 6 meter/second windspeed case was 300 meters. The line of agent produced by the release was 100 meters long and the median droplet diameter was slightly above 300 microns.

The peak liquid deposition from the attacks was approximately 3 grams/square meter for the 1.5 meter/second windspeed, 1 gram/square meter for the 3 meter/second windspeed, but only approximately 50 milligrams/square meter for the 6 meter/second windspeed.

The volatility of VX is so low that there is little vapor produced in the period of weeks to months of agent evaporation; therefore no agent concentration or dosage area coverage charts are necessary for this attack.

The casualty area coverage charts show the casualty potential of the liquid dissemination. The significant area coverage occurs over approximately 1 square kilometer in all three of the meteorological cases in MOPP 0, MOPP 2, and Mask only clothing configurations. In the case of no medical therapy, most of these casualties would be lethal. Appropriate use of Atropine & 2-PAM produces approximately a one order of magnitude reduction (or a 90 per cent reduction) in likely lethal area coverage without an overgarment, two orders of magnitude reduction (or a 99 per cent reduction) in likely lethal area coverage when the overgarment is worn as in MOPP 2 for the 1.5 or 3 meter/second windspeed cases, and a four order of magnitude reduction (or a 99.99 per cent reduction) in likely lethal area coverage when the overgarment is worn as in MOPP 4 for the 1.5 or 3 meter/second windspeed cases. Appropriate use of Atropine & 2-PAM completely removes the potential lethal areas (or a 99.99 per cent reduction) for the 6 meter/second windspeed case. Use of the pyridostigmine pretreatment reduces the likely lethal areas by more than one order of magnitude (or more than a 90 per cent reduction) for MOPP 0 cases and by four orders of magnitude (or a 99.99 per cent reduction).

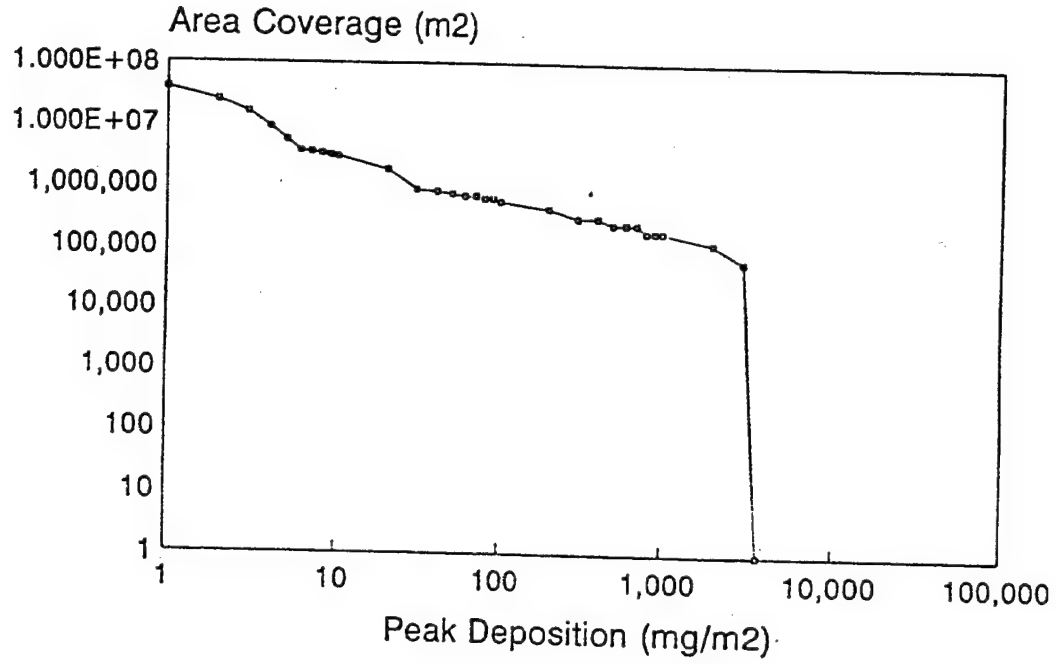
Tactical Ballistic Missile
Thickened VX



4oC (40oF)
1.5 m/sec
Stability E

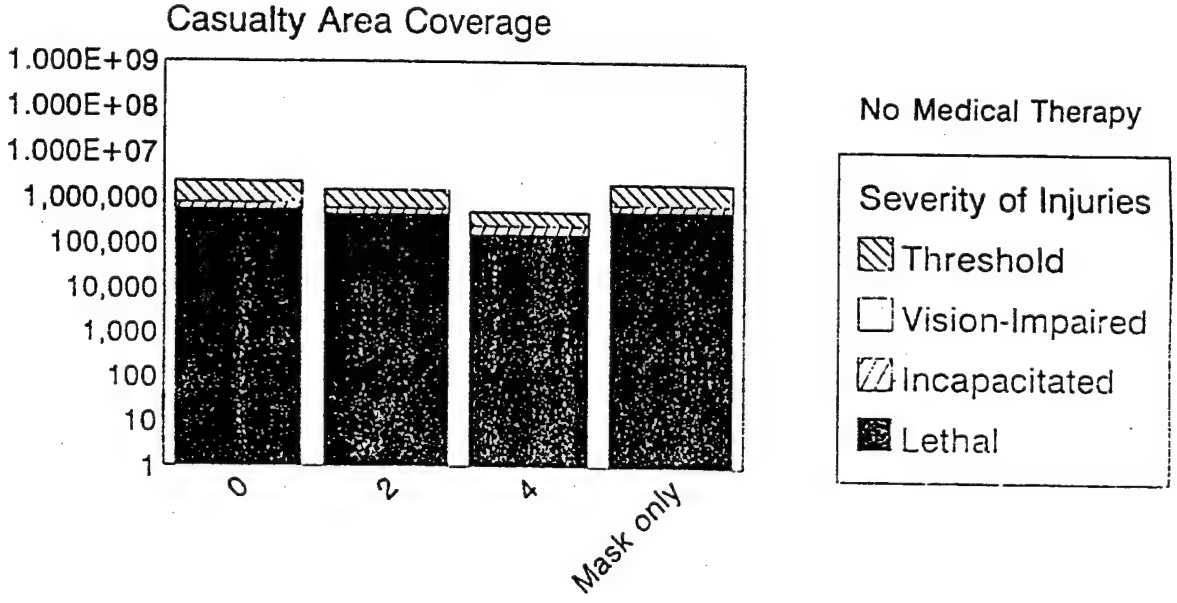
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened VX

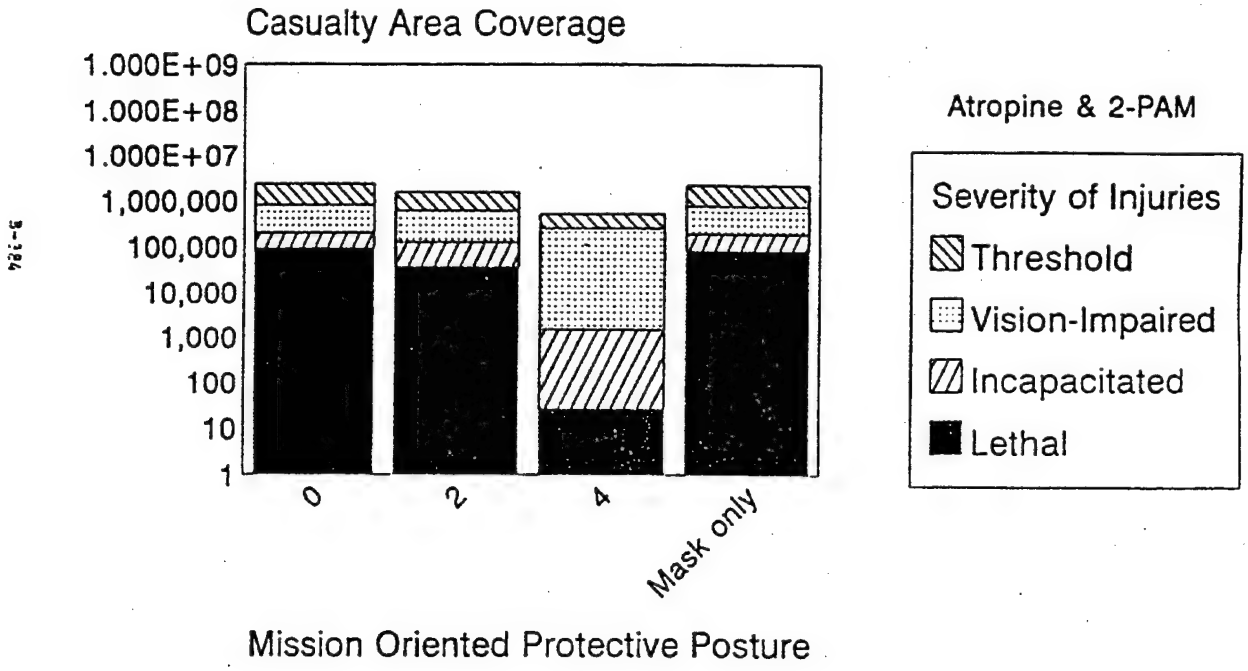


4°C (40°F), 1.5m/sec, stability E

Tactical Ballistic Missile
Thickened VX

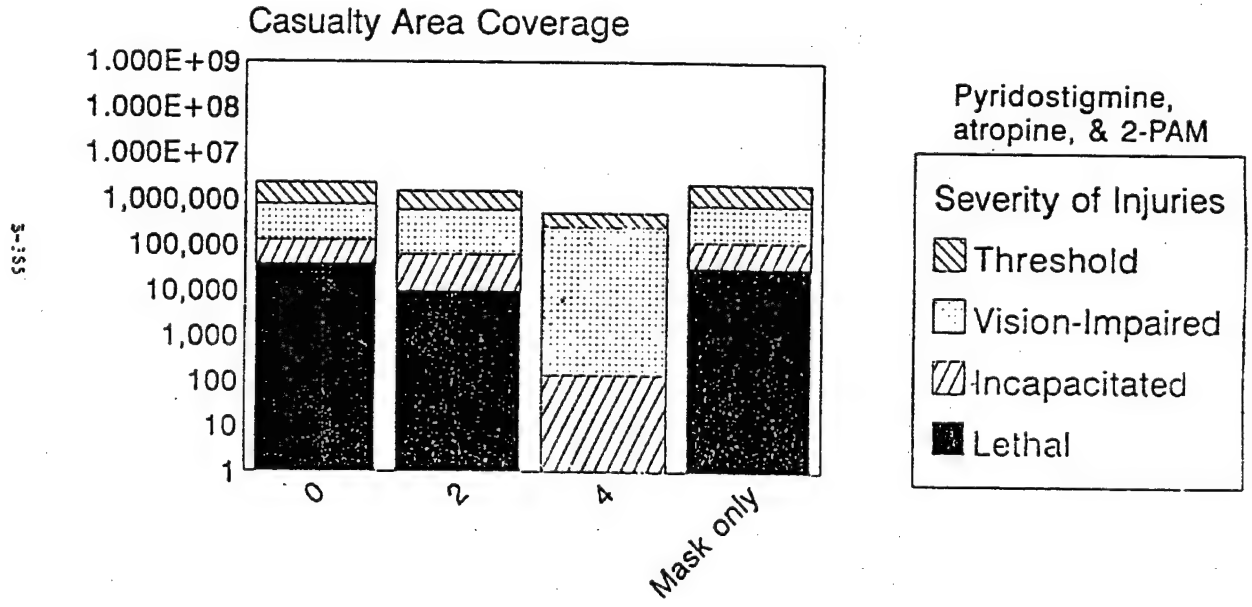


Tactical Ballistic Missile
Thickened VX



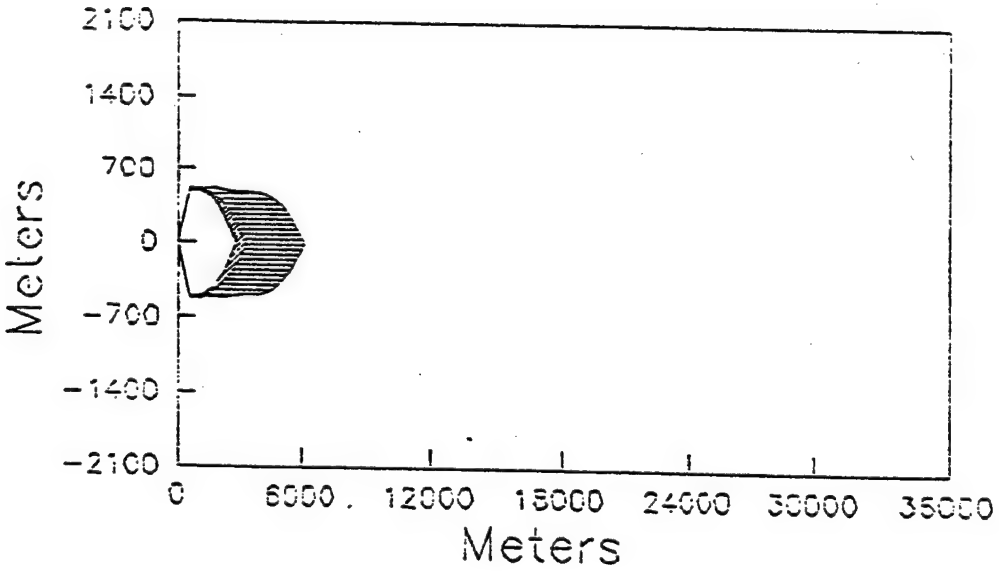
4°C (40°F), 3m/sec, Stability E

Tactical Ballistic Missile
Thickened VX



4°C (40°F), 3m/sec, Stability E

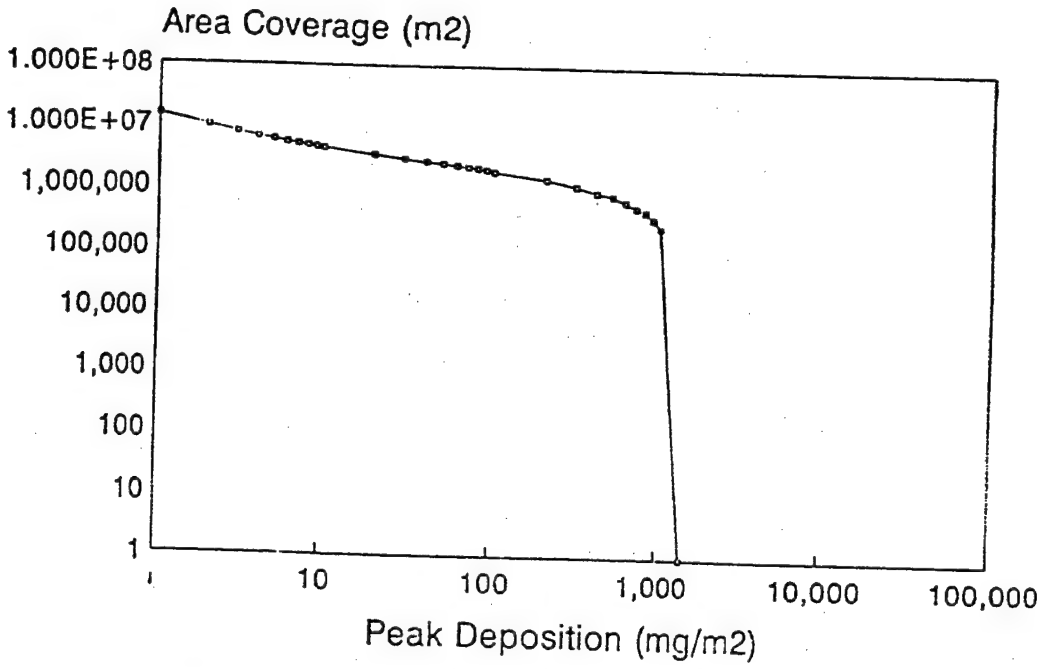
Tactical Ballistic Missile
Thickened VX



25oC (77oF)
3 m/sec
Stability D

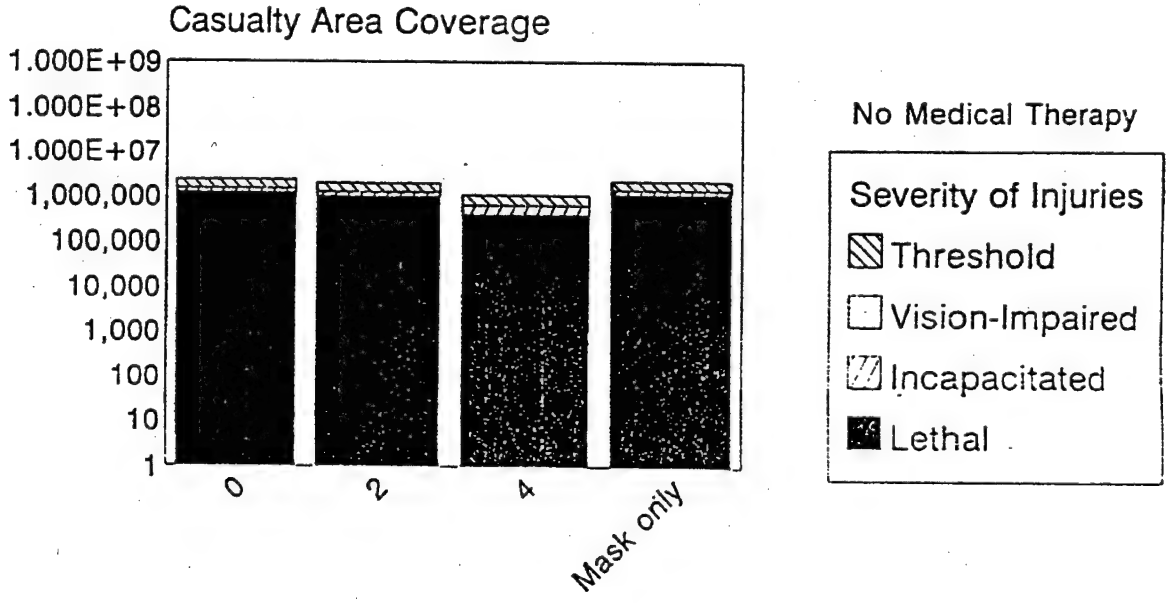
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened VX



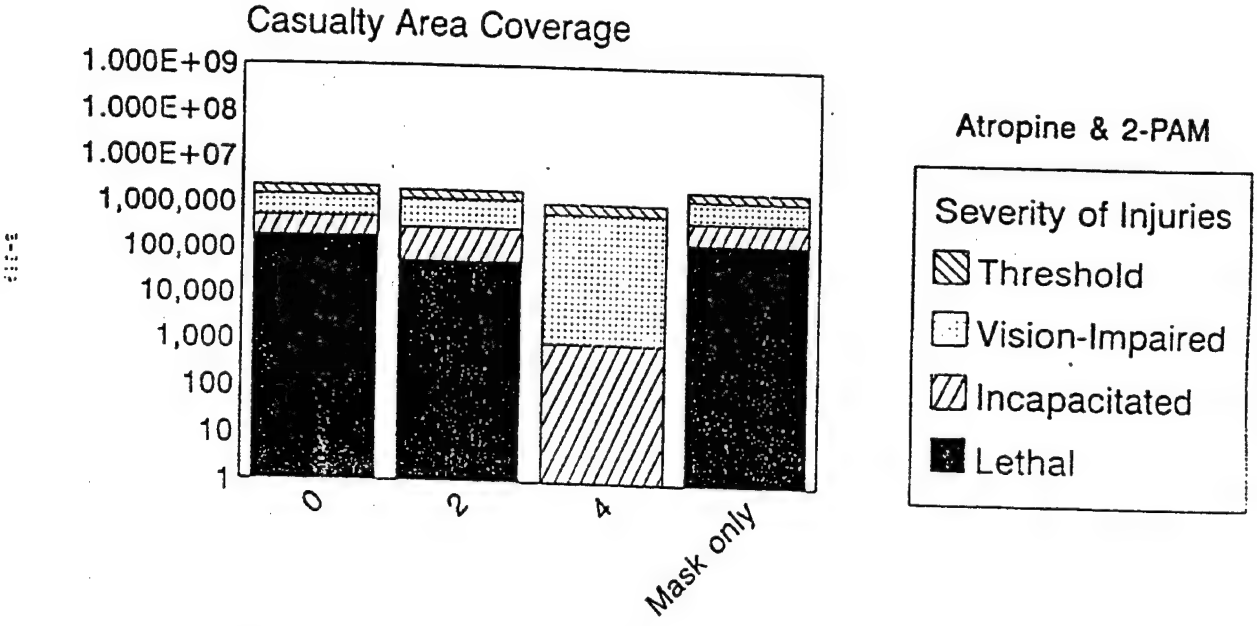
25°C (77°F), 3m/sec, stability D

Tactical Ballistic Missile
Thickened VX



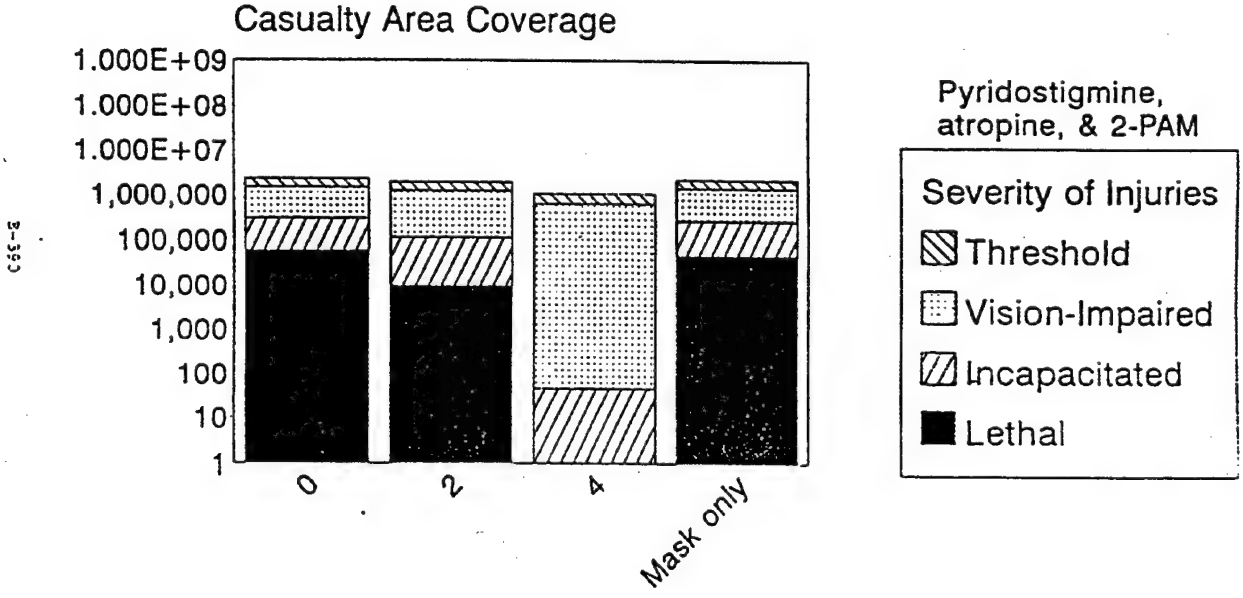
25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile
Thickened VX



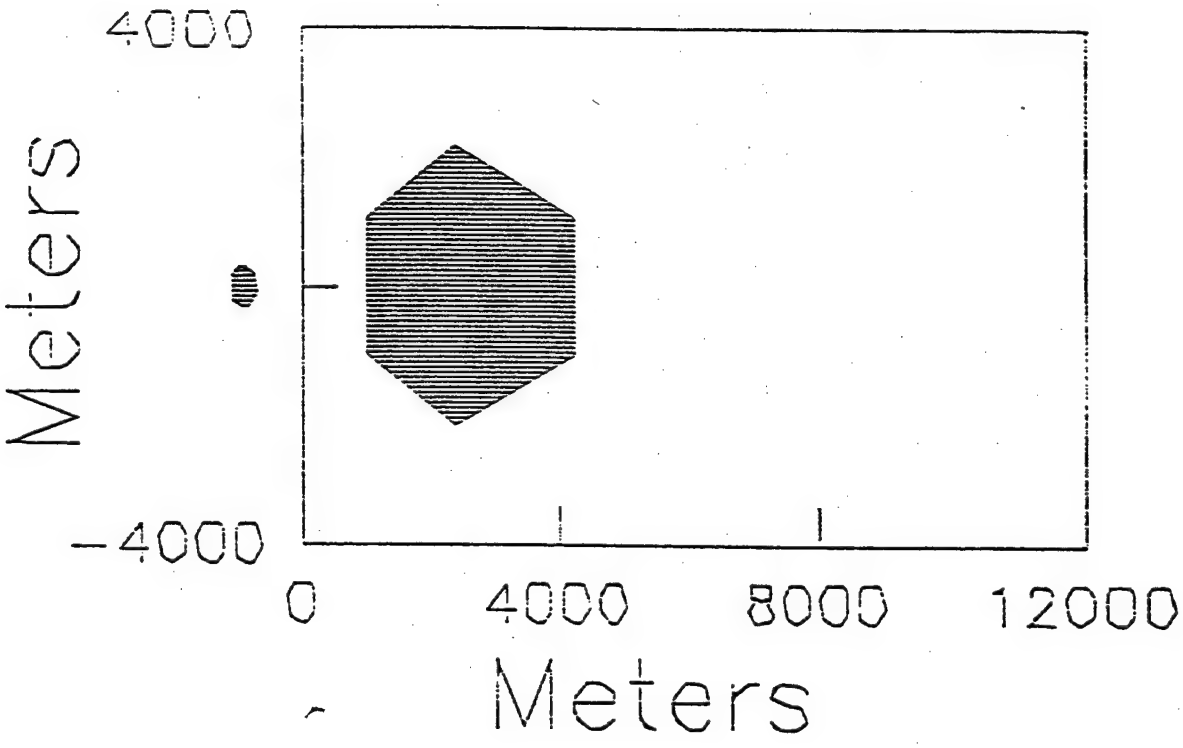
25°C (77°F), 3m/sec, Stability D

Tactical Ballistic Missile
Thickened VX



25°C (77°F), 3m/sec, Stability D

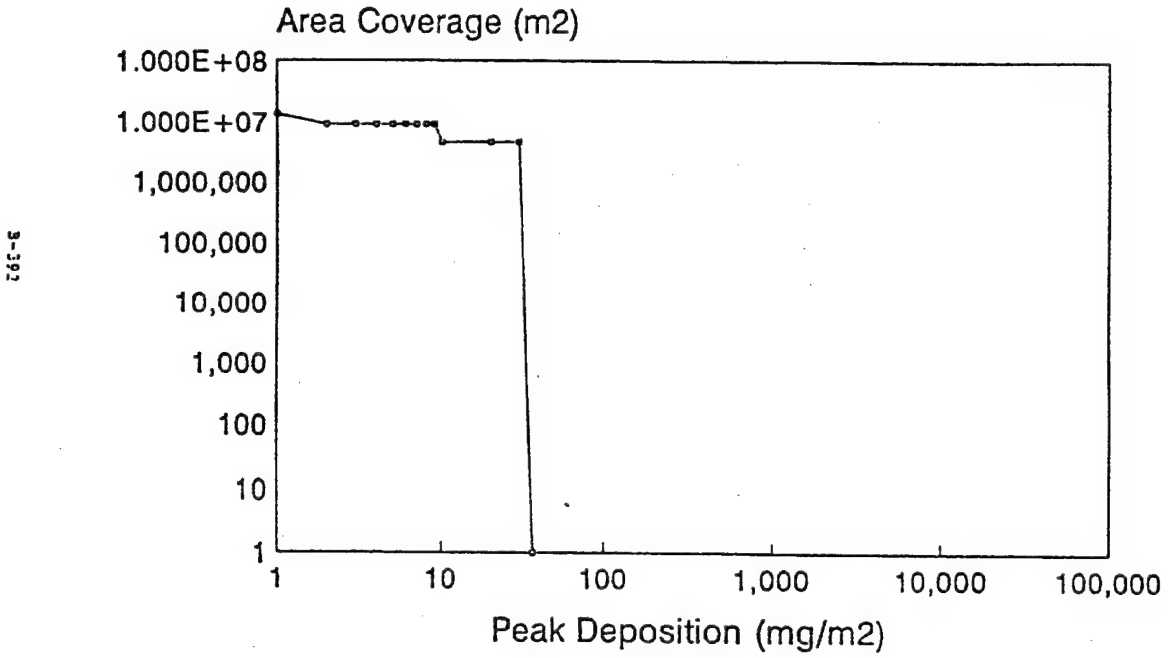
Tactical Ballistic Missile
Thickened VX



49oC (120oF)
6 m/sec
Stability B

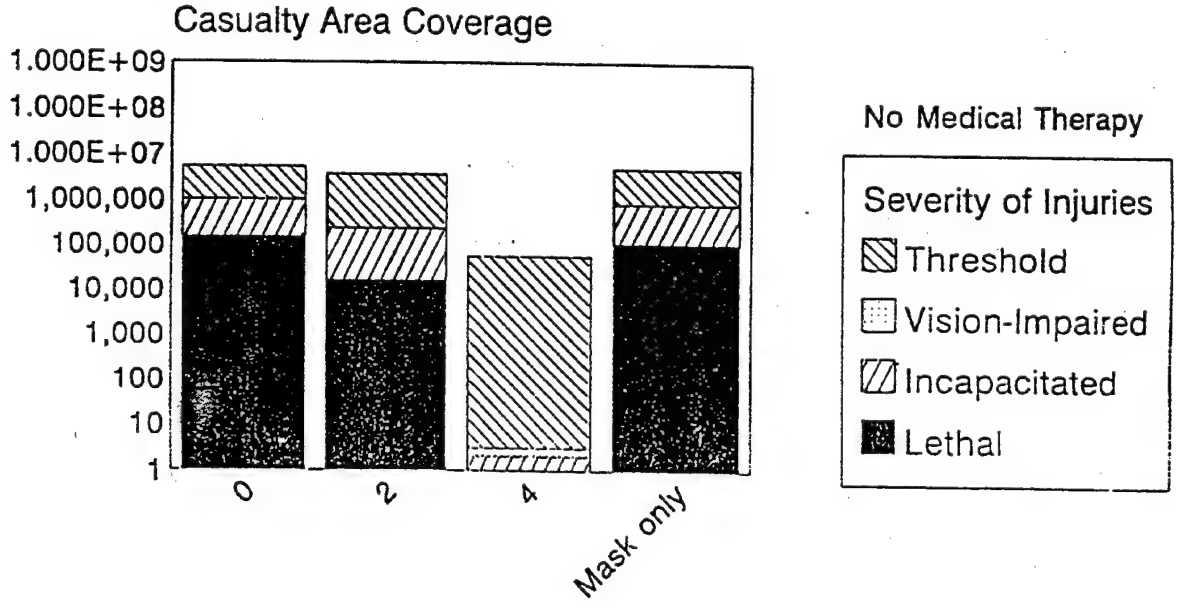
Visually Impaired
Incapacitated
Lethal

Tactical Ballistic Missile
Thickened VX

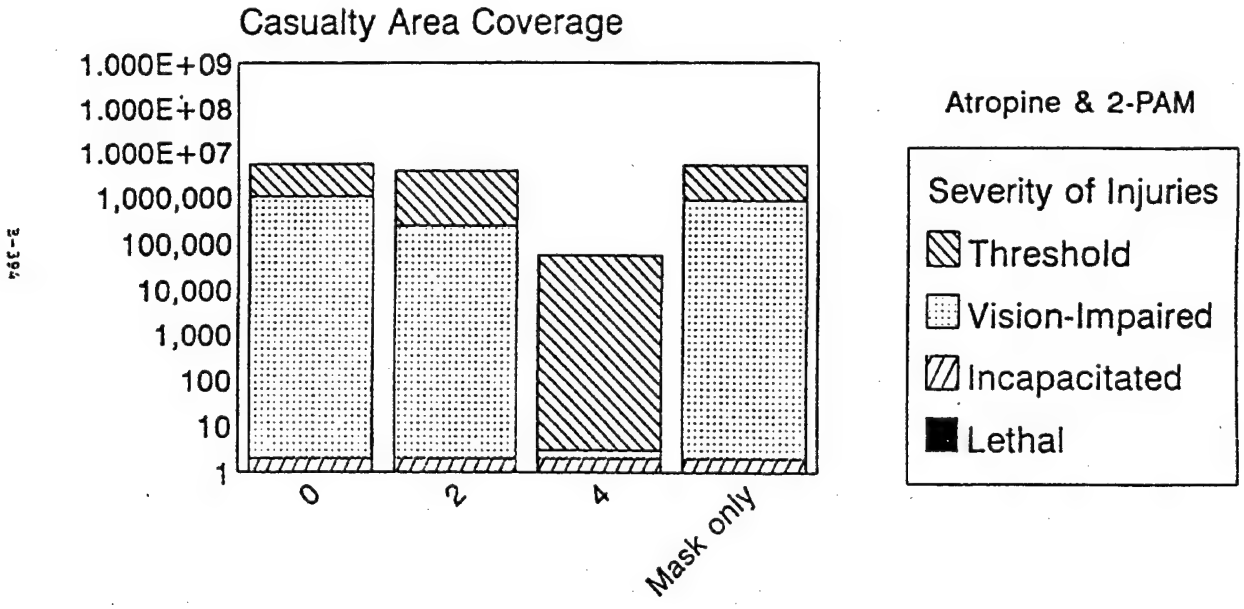


49°C (120°F), 6m/sec, stability B

Tactical Ballistic Missile
Thickened VX

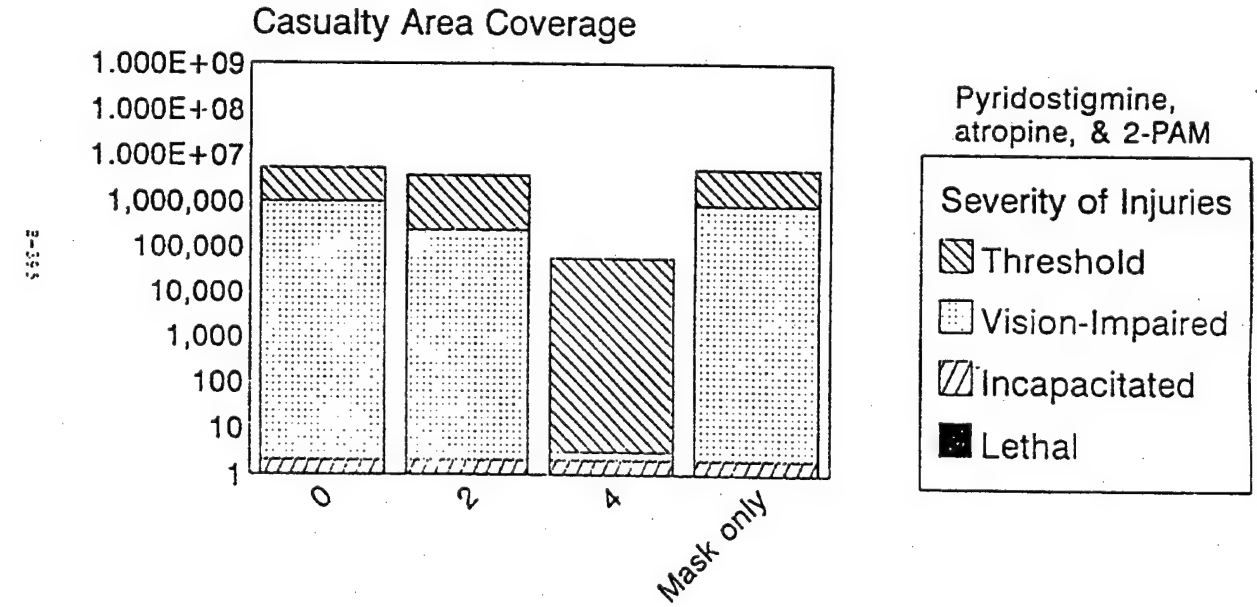


Tactical Ballistic Missile
Thickened VX



49°C (120°F), 6m/sec, Stability B

Tactical Ballistic Missile
Thickened VX



49°C (120°F), 6m/sec, Stability B

APPENDIX B

Evaporation of Neat and Thickened Chemical Agent from Short Grass Surfaces: Effects of Temperature and Wind Speed on Time Required for 90% Evaporation Source.† *[Note: Data assume 250-m droplets for neat agent and 1500-2500-m droplets for thickened agent.]*

†Selected tables and graphs from: McNally, R.E., M.M. Stark, J.M. Powers, Jr., and M.A. Sanzone (1992). *Worldwide Chemical/Biological Threat to U.S. Air Bases Vol. II: Agent Characteristics*, data taken from Appendix A. Science Applications International Corp., Joppa, Maryland.

Table 5. (U) Tabun (GA)				
	Evaporation Time (min) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	6461	2687	1545	888
0°C (32°F)	2148	893	513	295
10°C (50°F)	800	333	191	110
20°C (68°F)	329	136	78	45
30°C (86°F)	147	61	35	20
40°C (104°F)	70	29	16	9
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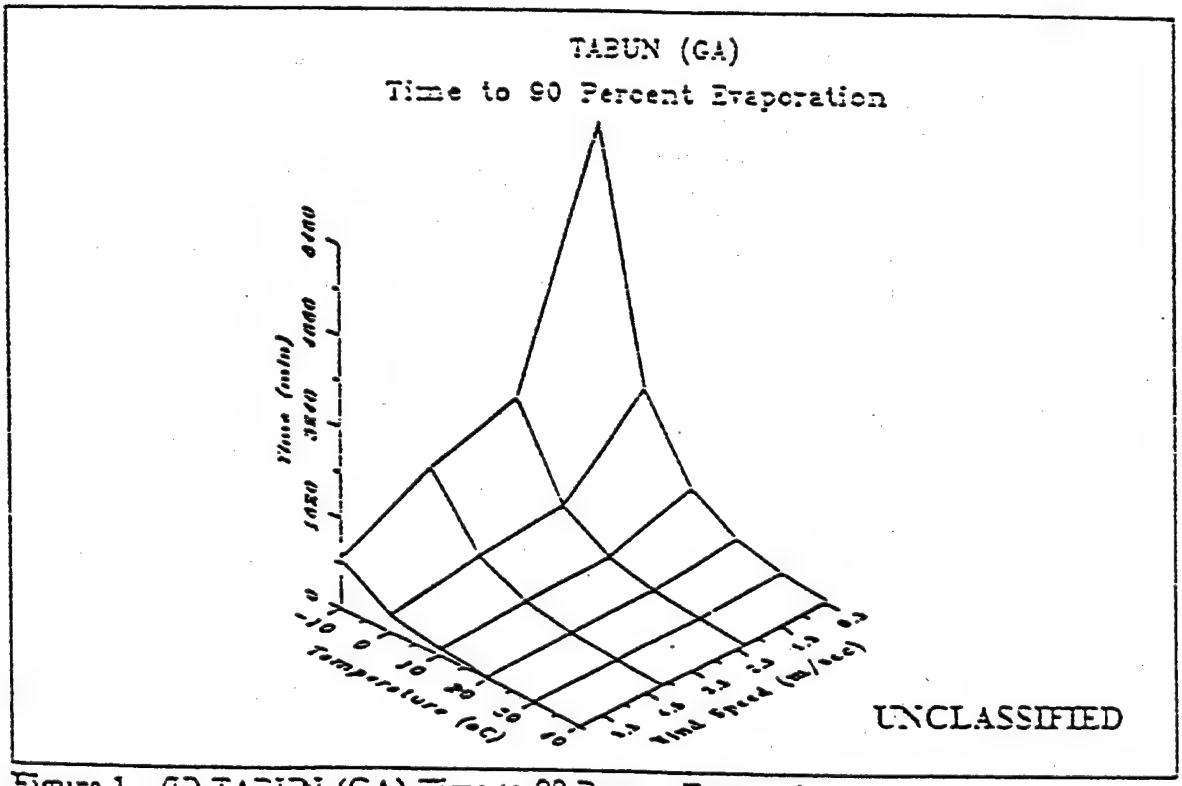

Figure 1. (U) TABUN (GA) Time to 90 Percent Evaporation.

Table 6. (U) Thickened Tabun (TGA)

Temperature	Evaporation Time (mins) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	40410	16809	9665	5557
0°C (32°F)	13435	5588	3213	1847
10°C (50°F)	5008	2083	1197	688
20°C (68°F)	2058	856	492	283
30°C (86°F)	920	382	220	126
40°C (104°F)	442	184	105	60

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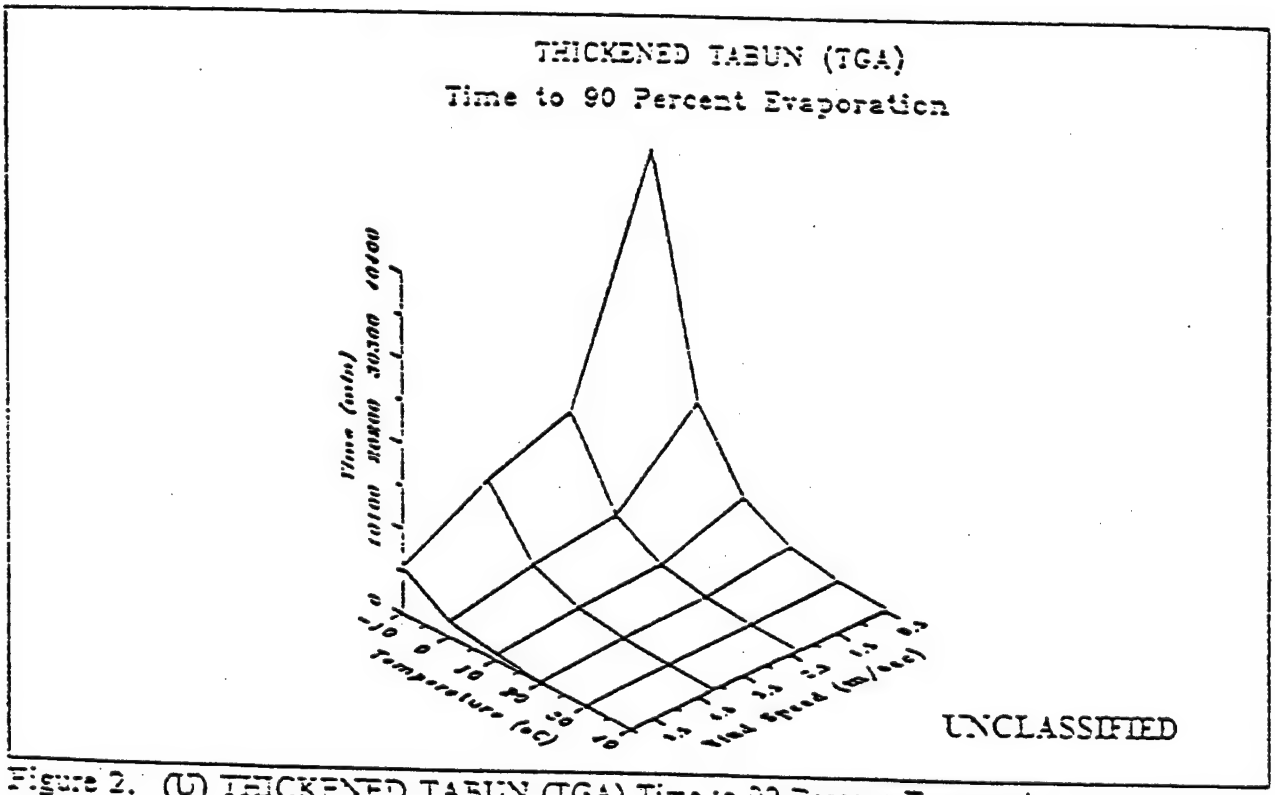

Figure 2. (U) THICKENED TABUN (TGA) Time to 90 Percent Evaporation.

Table 7. (U) Sarin (GB)				
	Evaporation Time (min) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	61	25	14	8
0°C (32°F)	29	12	6	3
10°C (50°F)	14	6	3	2
20°C (68°F)	7	3	1.9	1.1
30°C (86°F)	4	1.8	1.1	.6
40°C (104°F)	2	1.1	.6	.4
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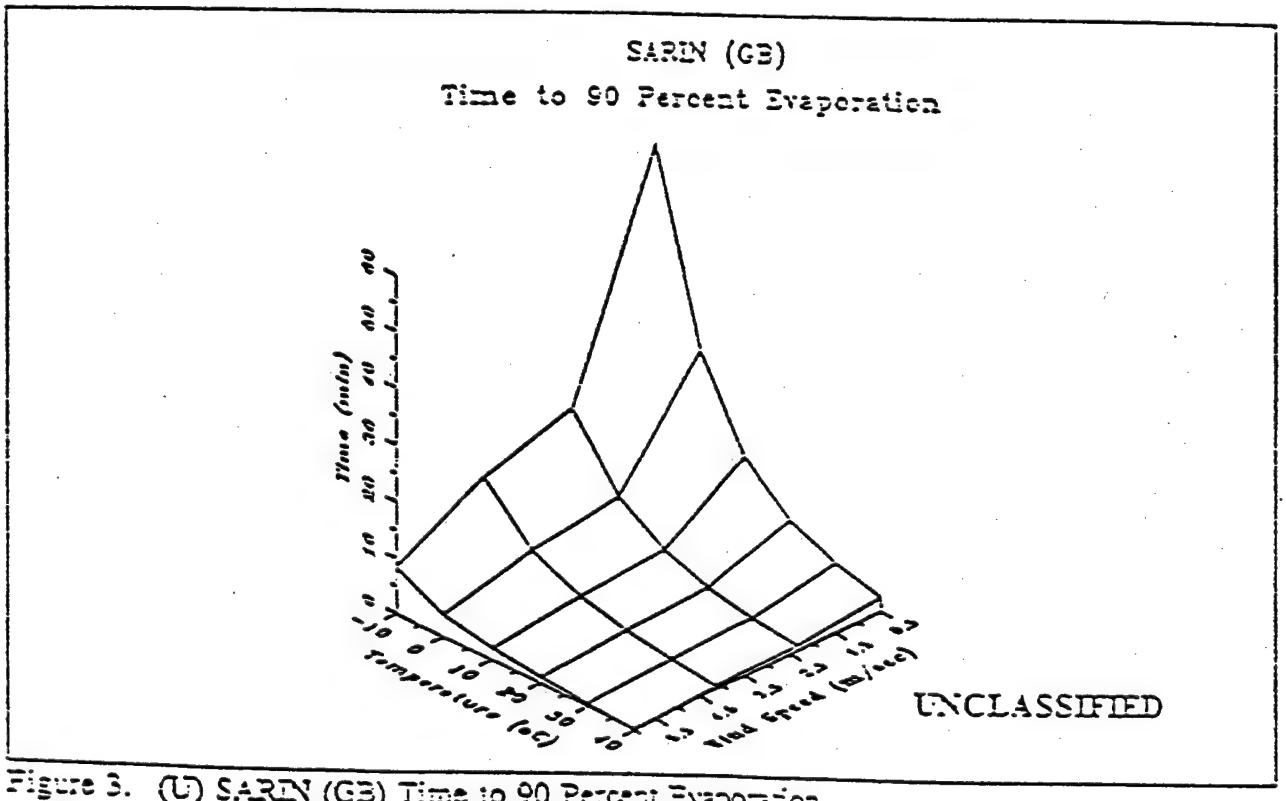

Figure 3. (U) SARIN (GB) Time to 90 Percent Evaporation.

Table 8. (U) Thickened Sarin (TGB)

Temperature	Evaporation Time (min) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	384	159	91	52
0°C (32°F)	181	75	43	24
10°C (50°F)	91	38	21	12
20°C (68°F)	48	20	11	6
30°C (86°F)	27	11	6	3
40°C (104°F)	16	6	3	2

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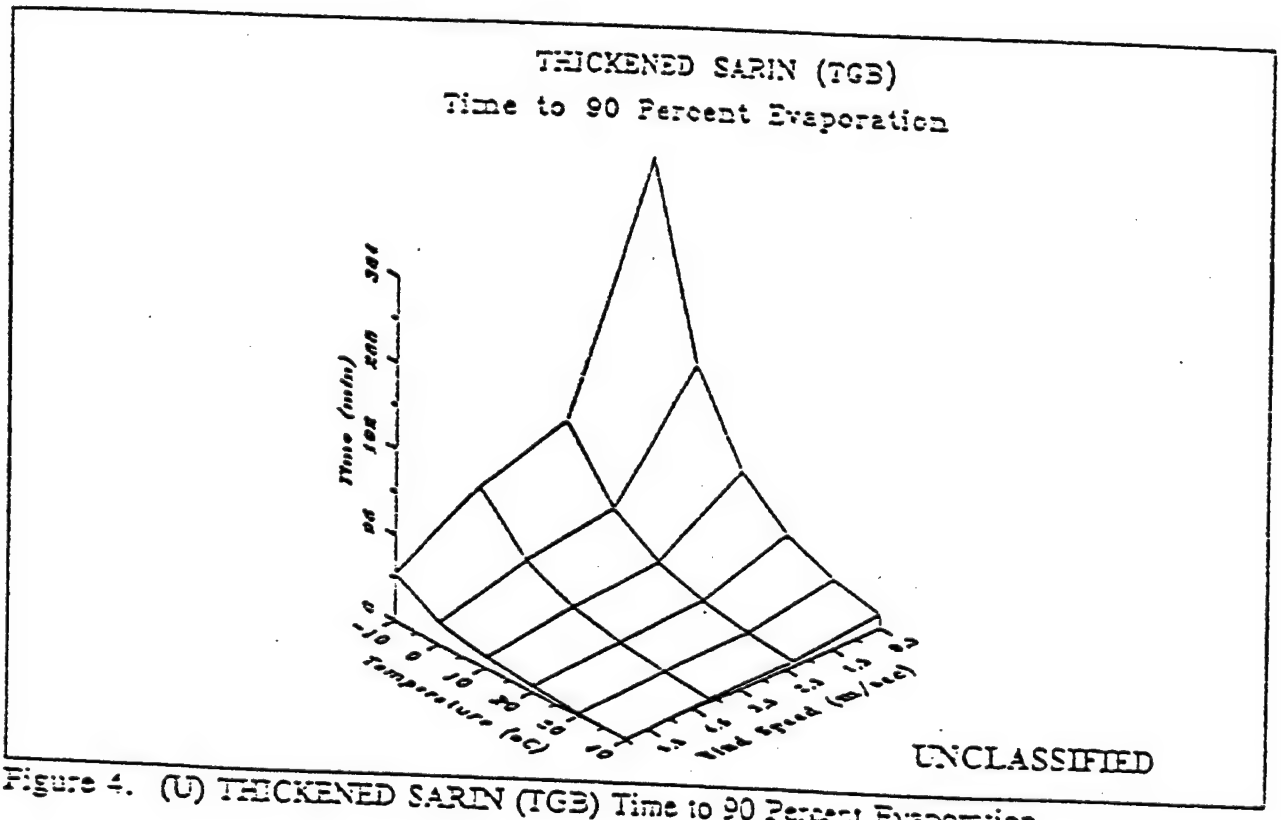

Figure 4. (U) THICKENED SARIN (TGB) Time to 90 Percent Evaporation.

Table 9. (U) Soman (GD)

Evaporation Time (min) for 90% Evaporation				
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	500	208	119	68
0°C (32°F)	204	84	48	28
10°C (50°F)	90	37	21	12
20°C (68°F)	42	17	10	5
30°C (86°F)	21	9	5	2
40°C (104°F)	11	4	2	1.6

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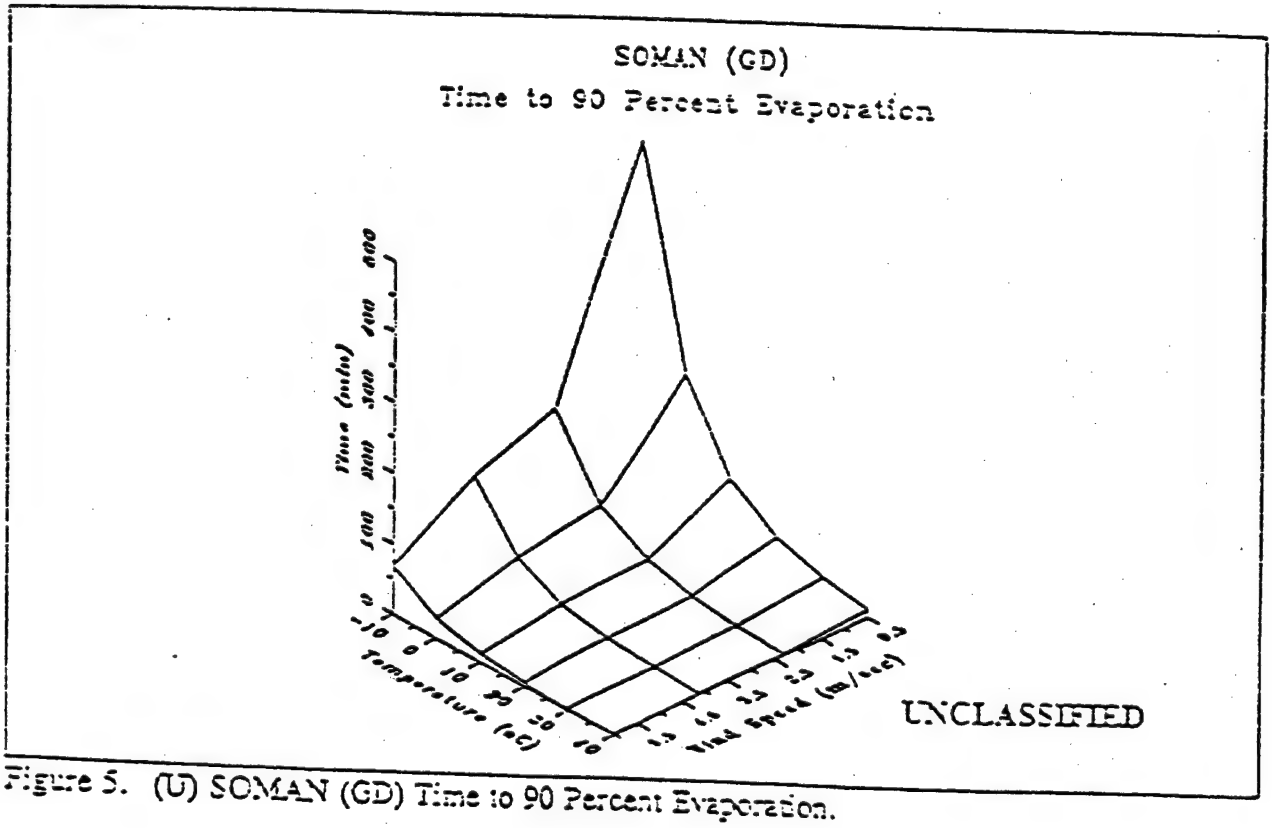

Figure 5. (U) SOMAN (GD) Time to 90 Percent Evaporation.

Table 10. (U) Thickened Soman (TGD)

Temperature	Evaporation Time (min) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	3131	1302	749	430
0°C (32°F)	1277	531	305	175
10°C (50°F)	565	235	135	77
20°C (68°F)	268	111	64	36
30°C (86°F)	135	56	32	18
40°C (104°F)	72	30	17	9

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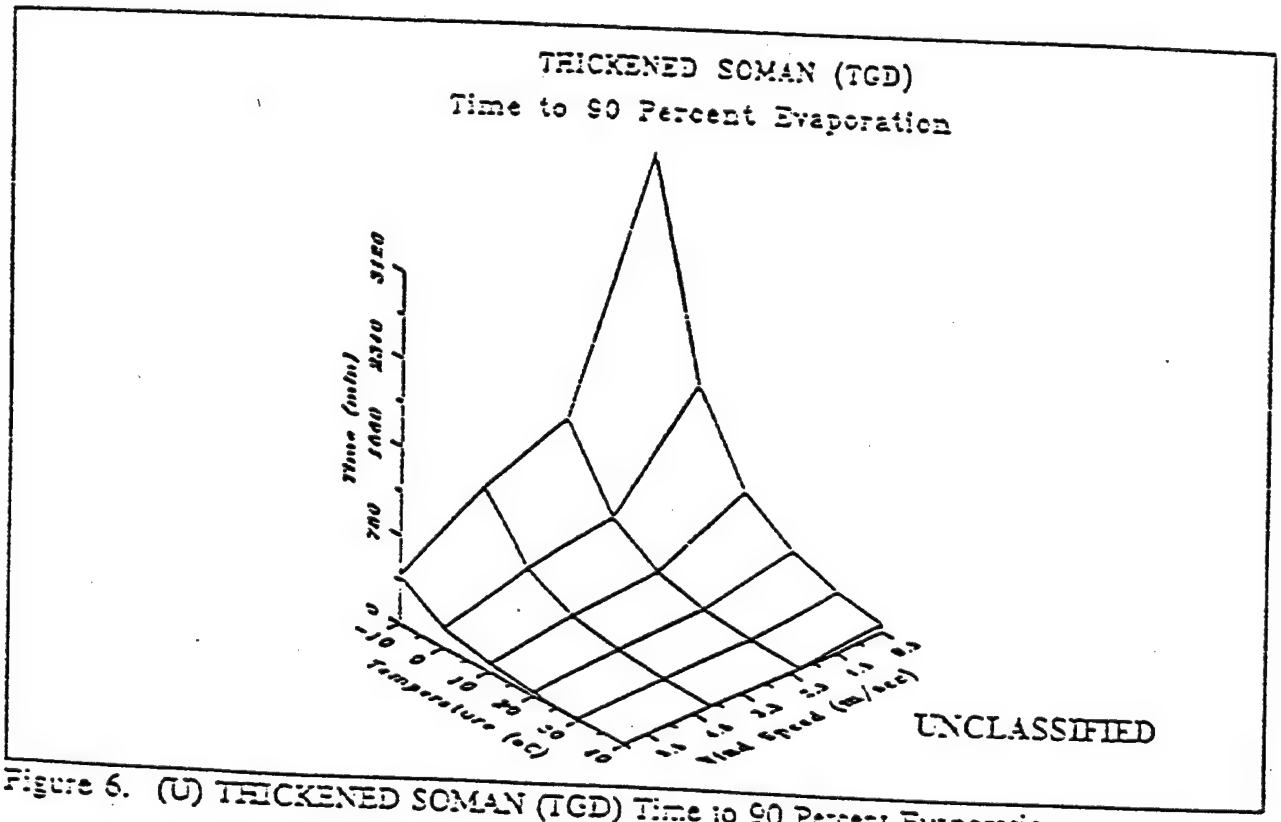

Figure 6. (U) THICKENED SOMAN (TGD) Time to 90 Percent Evaporation.

Table 11. (U) GF

Evaporation Time (min) for 90% Evaporation				
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	5769	2399	1379	793
0°C (32°F)	1778	739	425	244
10°C (50°F)	626	260	149	86
20°C (68°F)	246	102	59	33
30°C (86°F)	106	44	25	14
40°C (104°F)	50	20	12	6

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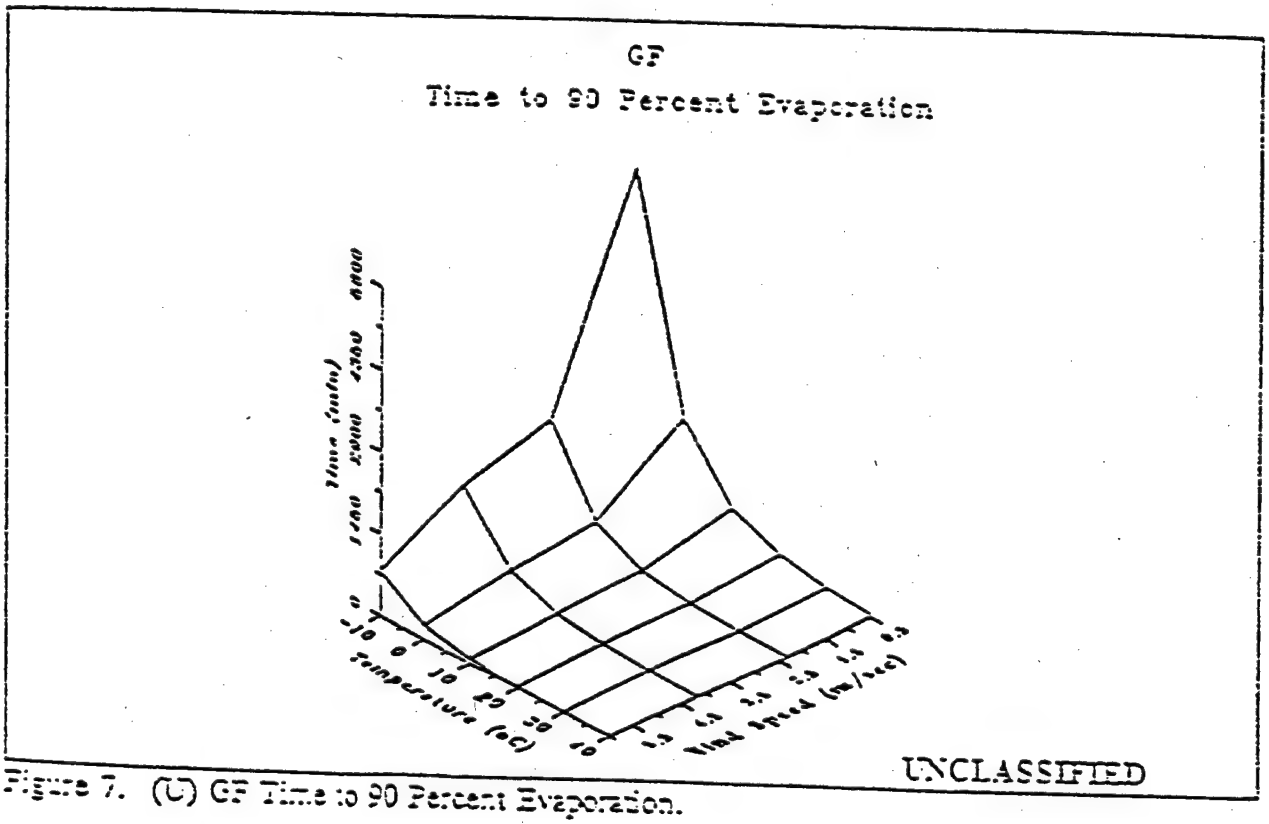

Figure 7. (U) GF Time to 90 Percent Evaporation.

Table 12. (U) Thickened GF

Temperature	Evaporation Time (min) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	36079	15008	8629	4961
0°C (32°F)	11124	4627	2660	1529
10°C (50°F)	3918	1629	937	538
20°C (68°F)	1543	642	369	212
30°C (86°F)	668	278	160	92
40°C (104°F)	314	130	75	43

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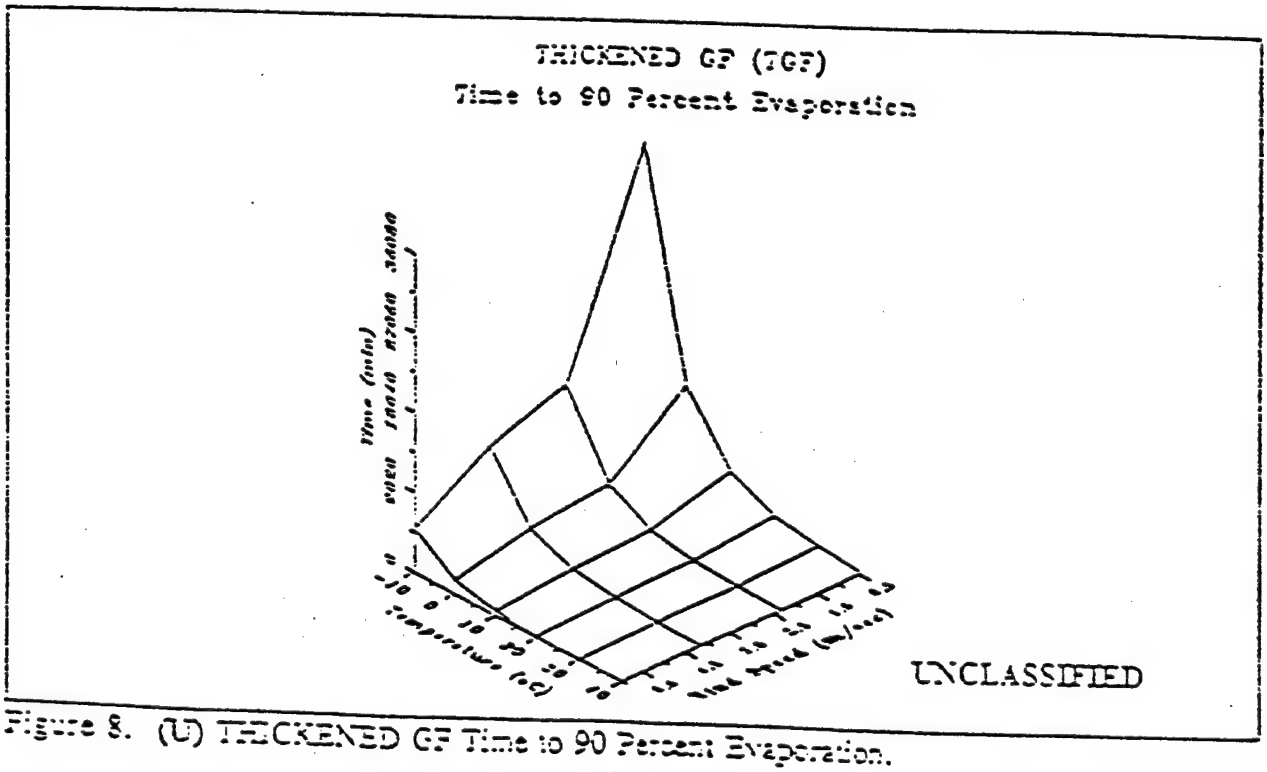

Figure 8. (U) THICKENED GF Time to 90 Percent Evaporation.

Table 13. (U) VX				
	Evaporation Time (days) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	901	375	215	124
0°C (32°F)	184	76	44	25
10°C (50°F)	44	18	10	6
20°C (68°F)	12	5	3	1.8
30°C (86°F)	4	1.7	1.0	.6
40°C (104°F)	1.5	.6	.4	.2
UNCLASSIFIED				

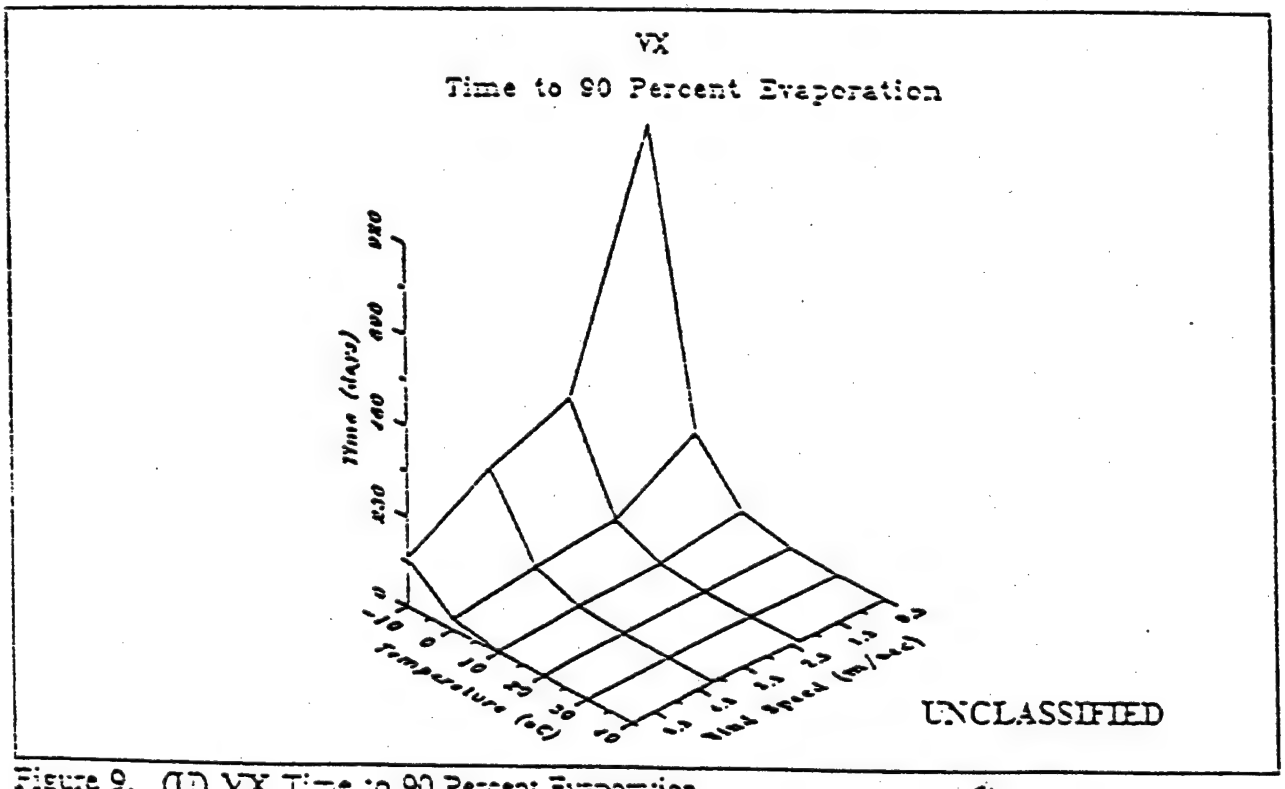

Figure 9. (U) VX Time to 90 Percent Evaporation.

Table 14. (U) Thickened VX (TVX)

Temperature	Evaporation Time (days) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	5639	2346	1348	775
0°C (32°F)	1151	478	275	158
10°C (50°F)	280	116	67	38
20°C (68°F)	79	32	18	10
30°C (86°F)	25	10	6	3
40°C (104°F)	9	3	2	1.3

UNCLASSIFIED

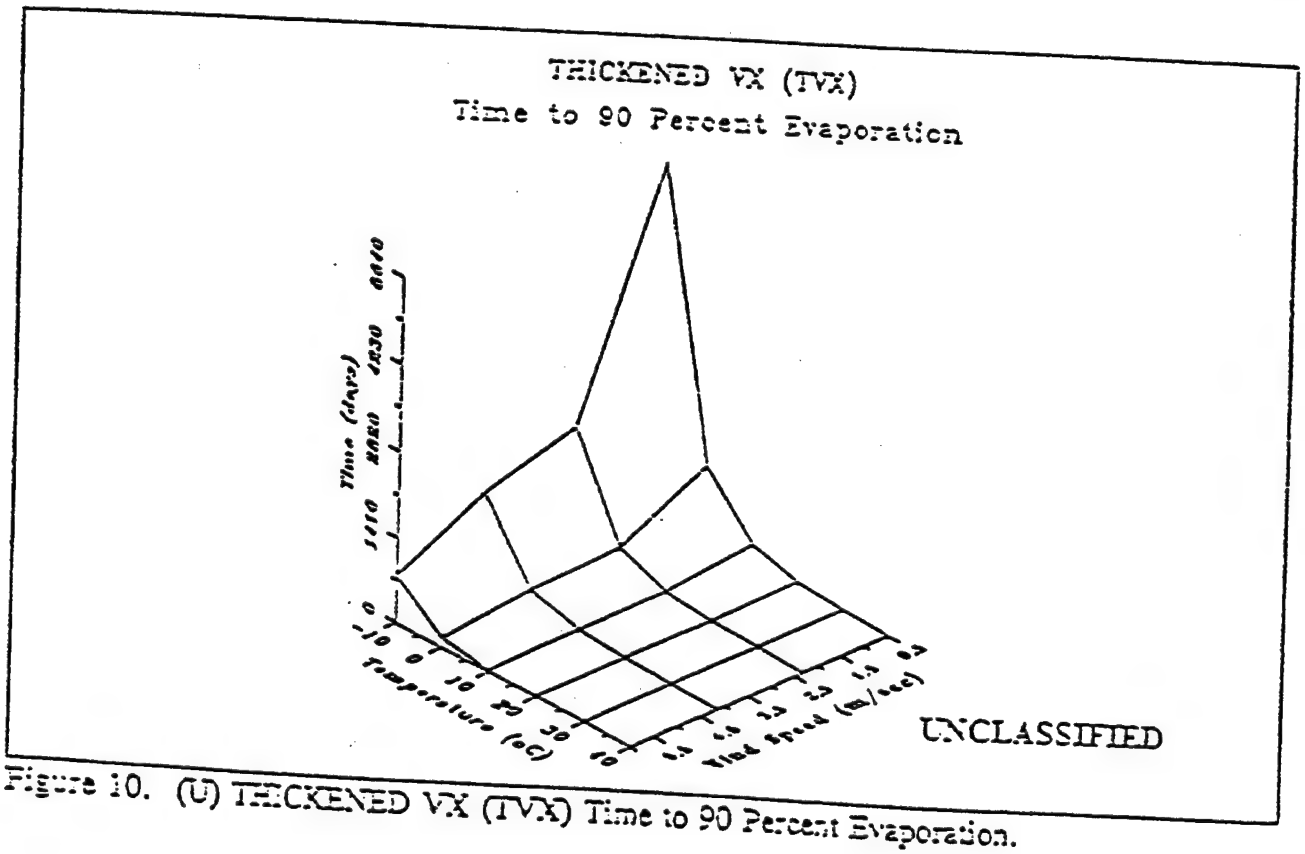

Figure 10. (U) THICKENED VX (TVX) Time to 90 Percent Evaporation.

Table 19. (U) Mustard (HD)

Temperature	Evaporation Time (min) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	3087	1284	738	424
0°C (32°F)	1094	455	261	150
10°C (50°F)	428	178	102	58
20°C (68°F)	182	75	43	25
30°C (86°F)	83	34	20	11
40°C (104°F)	41	17	9	5

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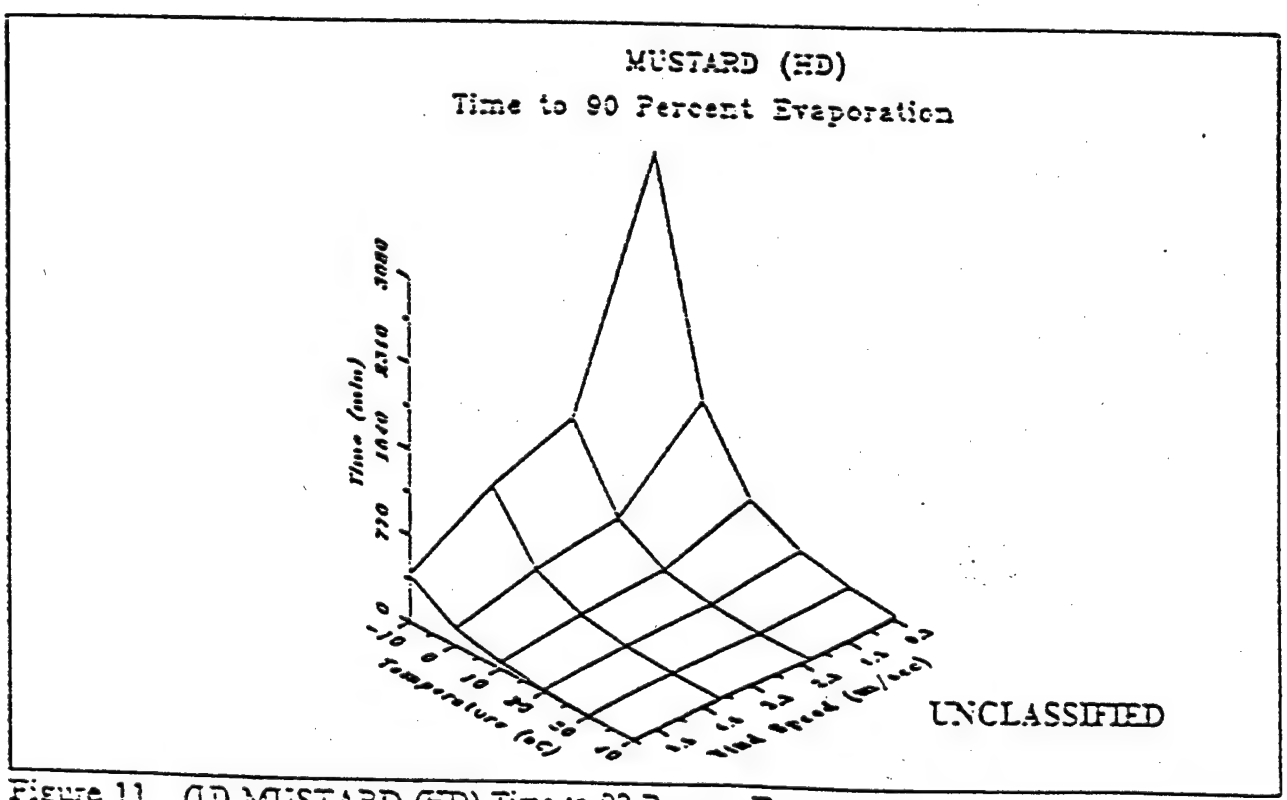

Figure 11. (U) MUSTARD (HD) Time to 90 Percent Evaporation

Table 20. (U) Thickened Mustard (THD)				
	Evaporation Time (min) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	19310	8032	4618	2655
0°C (32°F)	6844	2847	1637	941
10°C (50°F)	2678	1114	640	368
20°C (68°F)	1141	474	273	157
30°C (86°F)	524	218	125	72
40°C (104°F)	257	106	61	35
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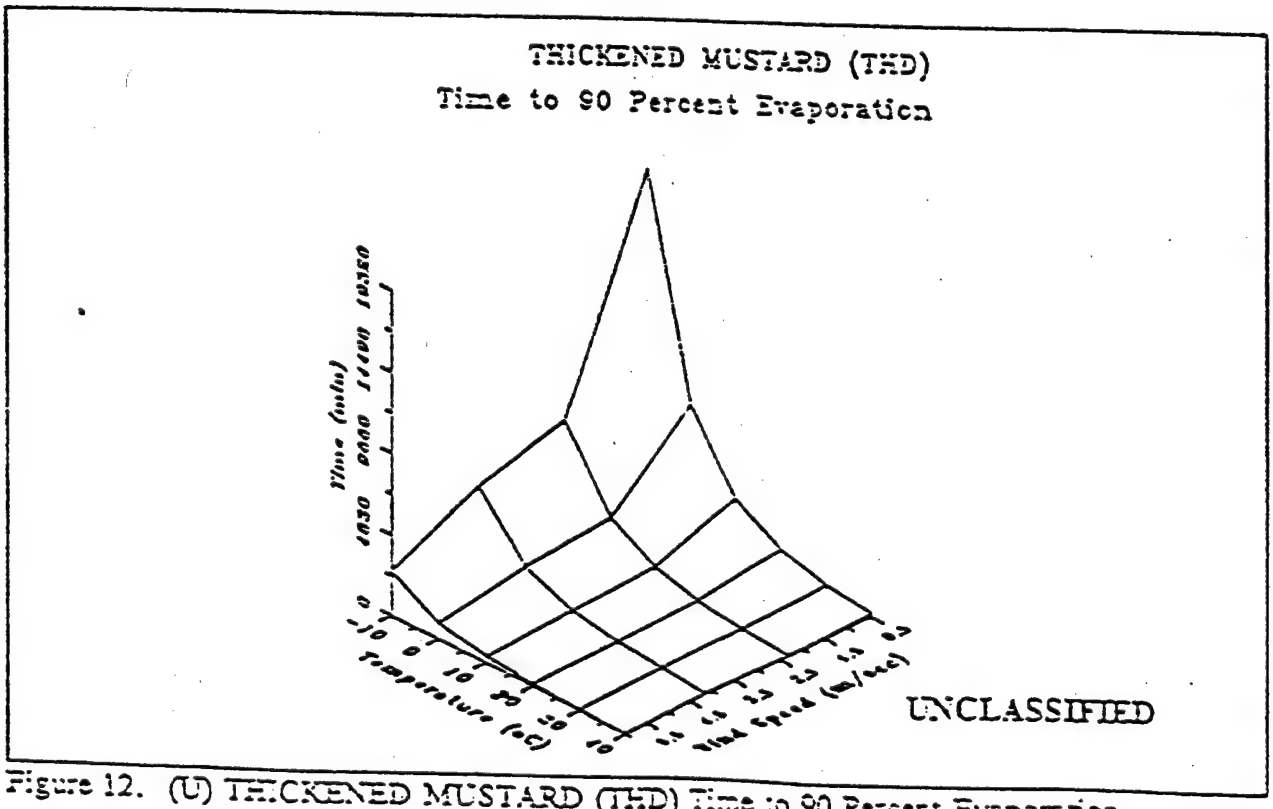

Figure 12. (U) THICKENED MUSTARD (THD) Time to 90 Percent Evaporation.

Table 21. (U) Lewisite (L)

Temperature	Evaporation Time (min) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	739	307	176	101
0°C (32°F)	276	114	66	38
10°C (50°F)	112	46	26	15
20°C (68°F)	49	20	11	6
30°C (86°F)	23	9	5	3
40°C (104°F)	11	4	2	1.6

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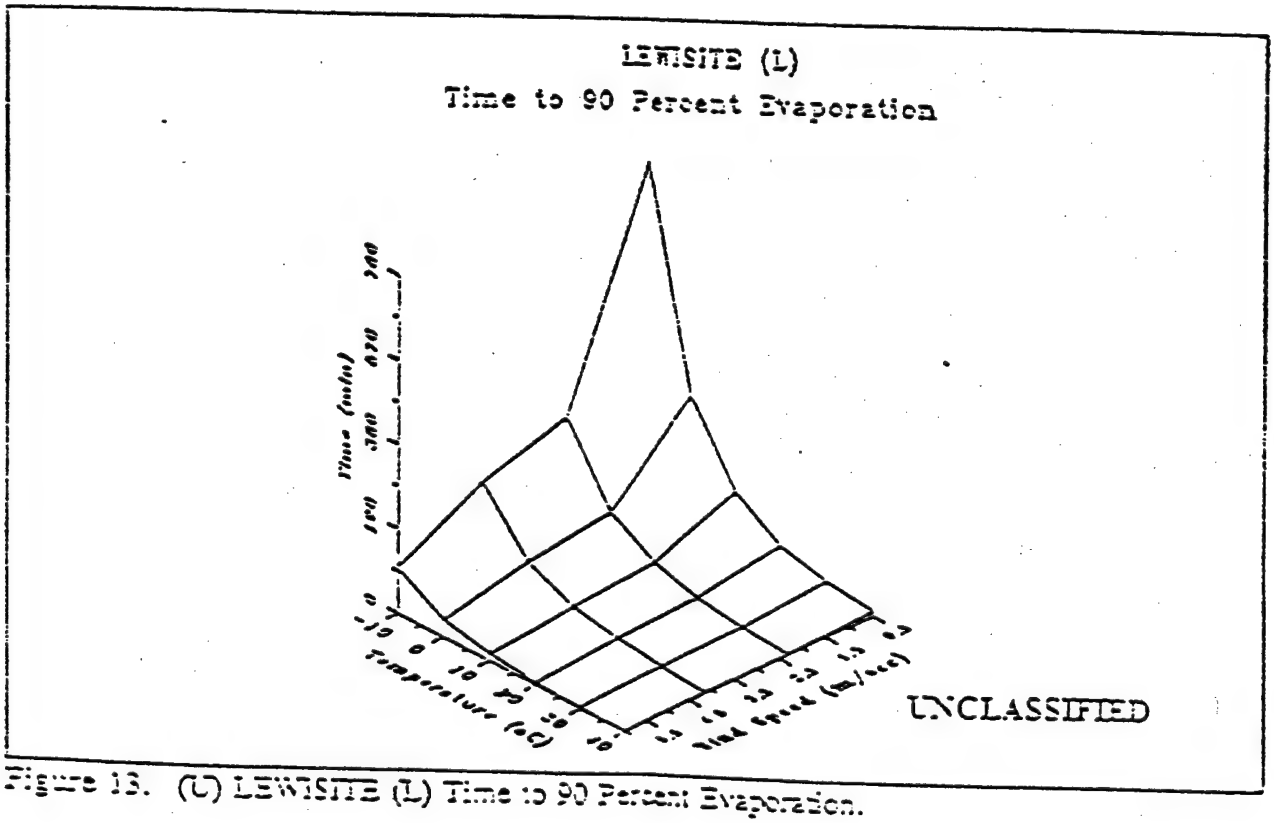

Figure 13. (U) LEWISITE (L) Time to 90 Percent Evaporation.

Table 22. (U) Thickened Lewisite (TL)				
	Evaporation Time (min) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	4626	1924	1106	656
0°C (32°F)	1728	718	413	237
10°C (50°F)	704	293	168	96
20°C (68°F)	310	128	74	42
30°C (86°F)	145	60	34	20
40°C (104°F)	72	30	17	10
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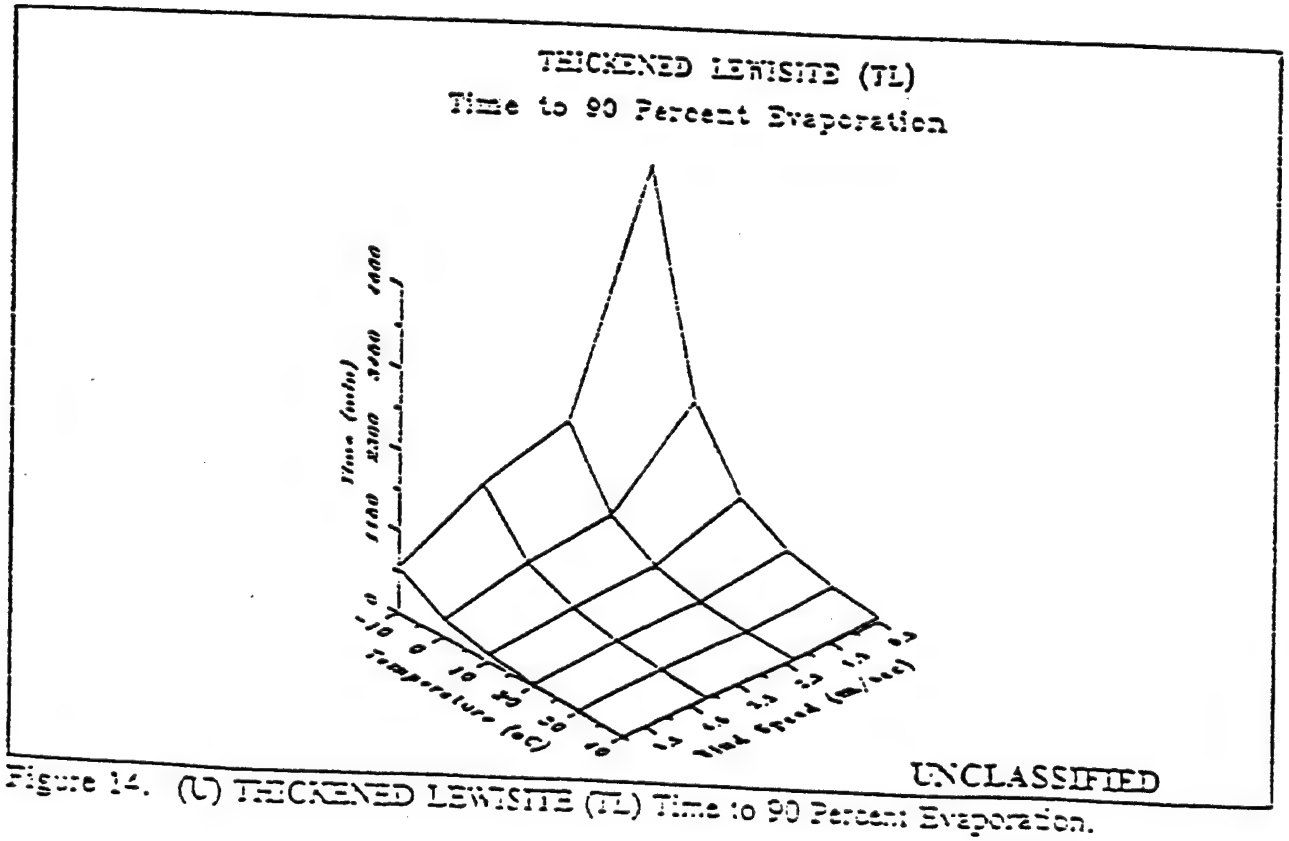

Figure 14. (U) THICKENED LEWISITE (TL) Time to 90 Percent Evaporation.

Table 23. (U) Phosgene Oxime (CX)				
	Evaporation Time (minutes) for 90% Evaporation			
Temperature	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	64	26	15	8
0°C (32°F)	28	11	6	3
10°C (50°F)	13	5	3	1.8
20°C (68°F)	6	2	1.5	.9
30°C (86°F)	3	1.4	.8	.5
40°C (140°F)	1.8	.7	.4	.3
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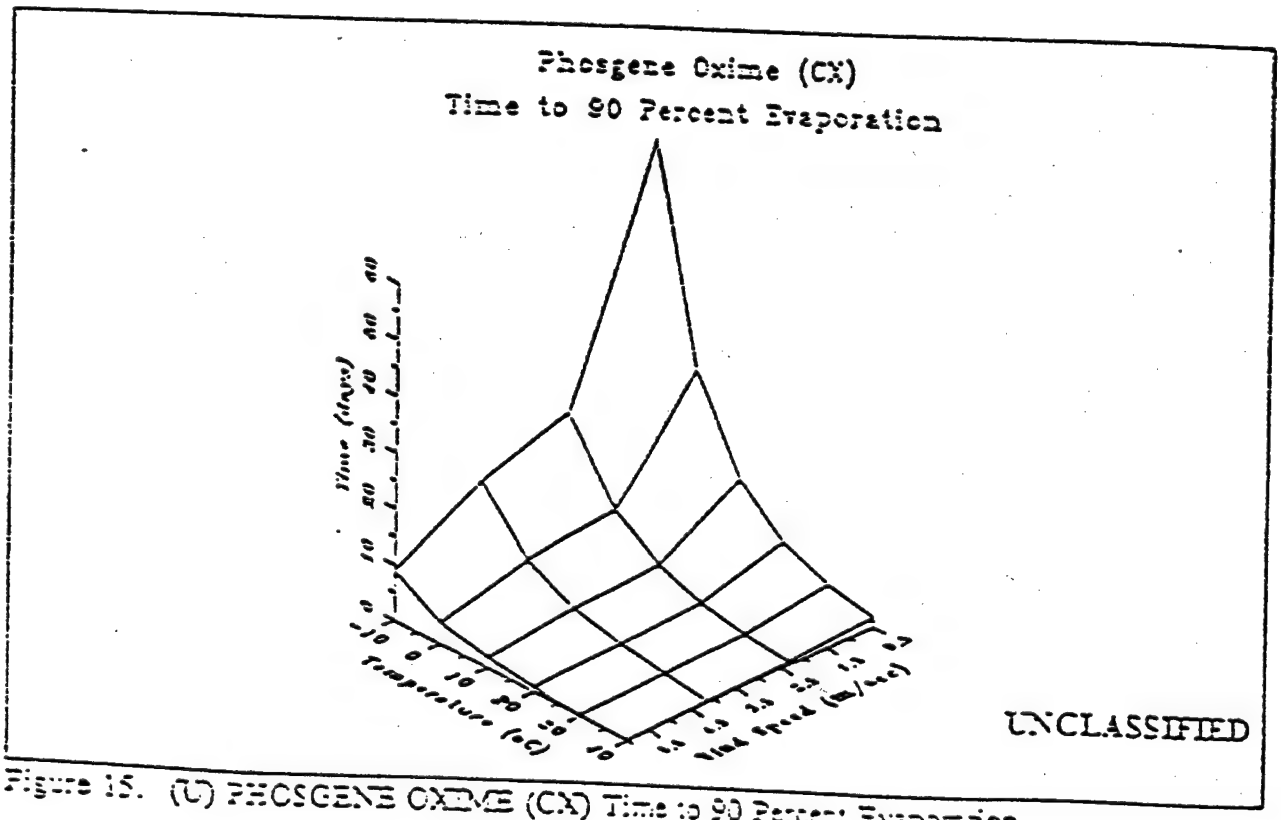

Figure 15. (U) PHOSGENE OXIME (CX) Time to 90 Percent Evaporation.

Table 24. (U) Hydrogen Cyanide (AC)

Temperature	Evaporation Time (sec) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	31	13	7	4
0°C (32°F)	20	8	5	2
10°C (50°F)	14	5	3	2
20°C (68°F)	10	4	2	1.4
30°C (86°F)	7	3	1.7	1
40°C (140°F)	5	2	1.3	.7

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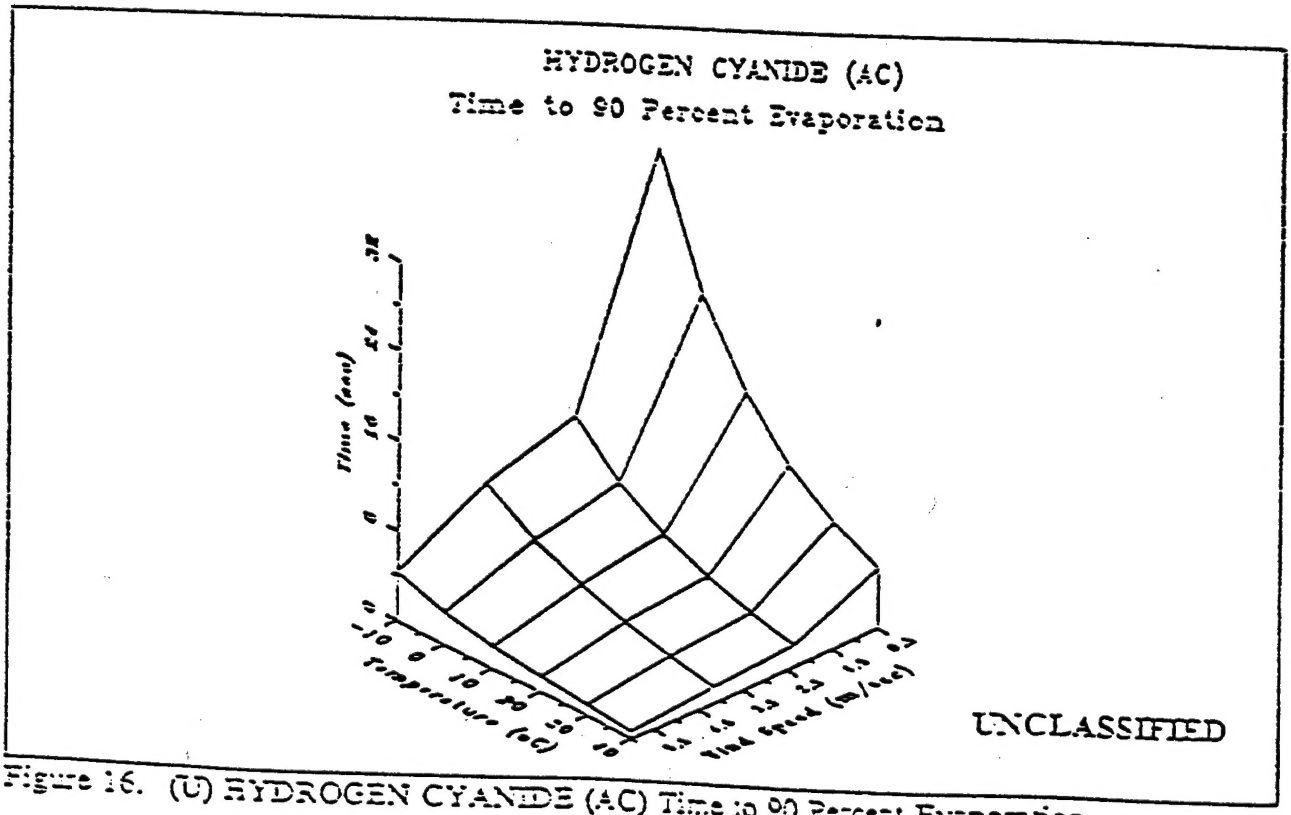

Figure 16. (U) HYDROGEN CYANIDE (AC) Time to 90 Percent Evaporation.

Table 25. (U) Cyanogen Chloride (CK)

Temperature	Evaporation Time (sec) for 90% Evaporation			
	.5 m/sec windspeed	1.5 m/sec windspeed	3 m/sec windspeed	6 m/sec windspeed
-10°C (14°F)	8	3	2	1.2
0°C (32°F)	5	2	1.4	.8
10°C (50°F)	4	1.7	1.0	.6
20°C (68°F)	2	1.2	.7	.4
30°C (86°F)	2	.9	.5	.3
40°C (140°F)	1.6	.7	.4	.2

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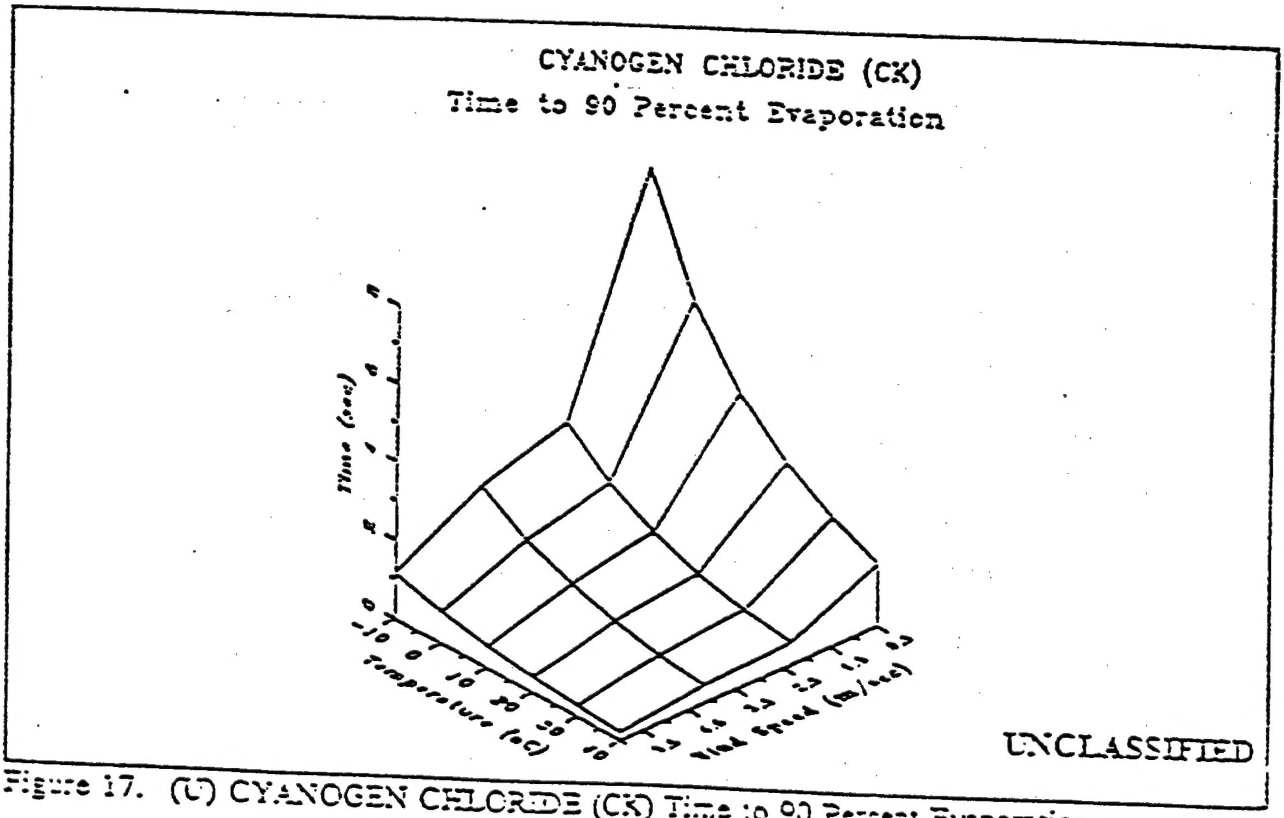

Figure 17. (U) CYANOGEN CHLORIDE (CK) Time to 90 Percent Evaporation.

APPENDIX C

Individuals and Organizations Contacted for Information Relating to Implications of the Decontamination Procedure on Embalming and Cosmetic Restoration

CONTACTED INDIVIDUALS AND ORGANIZATIONS

Rocky Bradford	Technical Representative, CTP Inc., Greenville, Illinois[†]
Dwayne Flowers	Sales Manager, CTP Inc., Greenville, Illinois
William Hearn	Dade County Medical Examiner's Office Miami, Florida
Robert Mayer	Licensed Embalmer, textbook author, Pittsburgh Institute of Mortuary Science, Pittsburgh, Pennsylvania
Eugene Ogrodnik	Dean, Pittsburgh Institute of Mortuary Science, Pittsburgh, Pennsylvania
Chris Robinson	Technical Representative, Dodge Chemical Company, Cambridge, Massachusetts
Roger Walker	Licensed Embalmer, Pittsburgh Institute of Mortuary Science, Pittsburgh, Pennsylvania
Margaret Wells	Chemist, Dodge Chemical Company, Cambridge, Massachusetts

[†]CTP Inc. distributes a product called Klorman that is marketed for use as a disinfectant in the embalmer's preparation room. The product, which bubbles chlorine over solid calcium hypochlorite (up to 600 ppm) at pH 9 in a water supply. Its intended use is for flushing of the embalming table. According to R. Bradford, the Israeli Defense Forces have been investigating use of Klorman for decontamination of human remains exposed to biological warfare agents.